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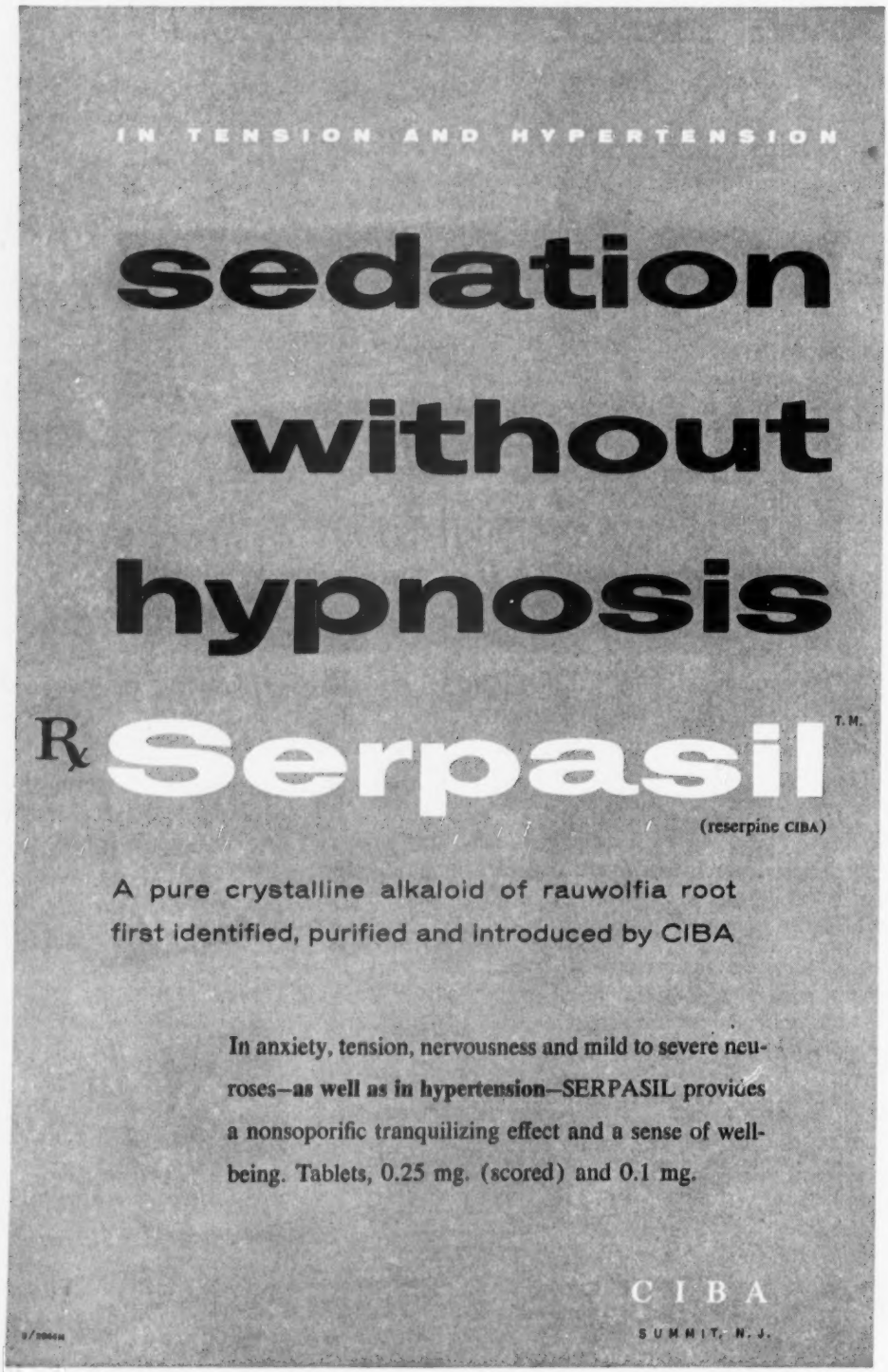
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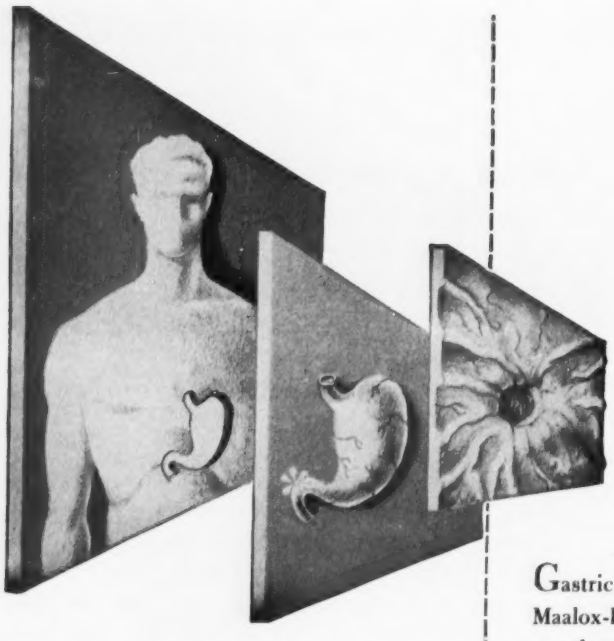

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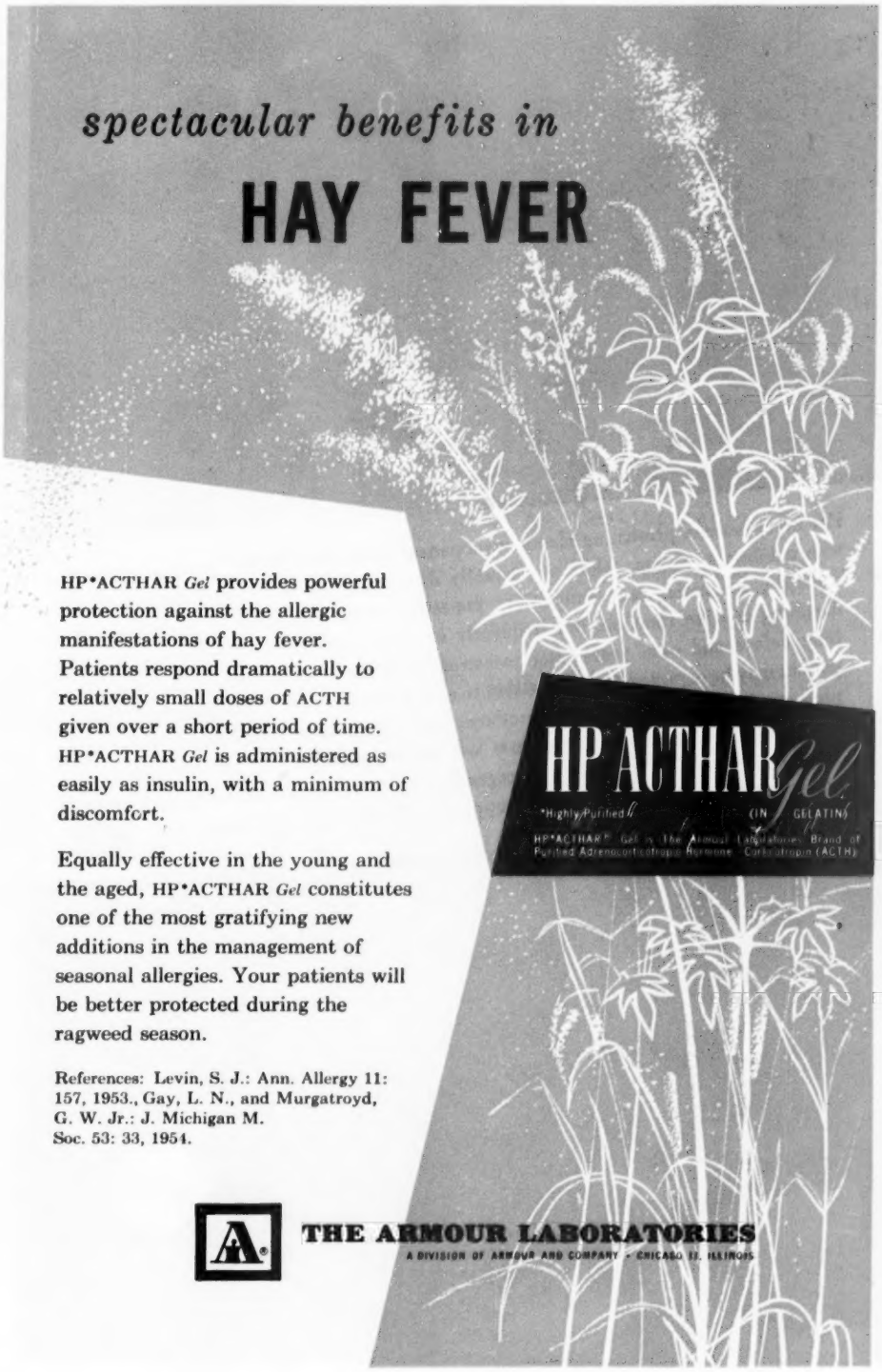
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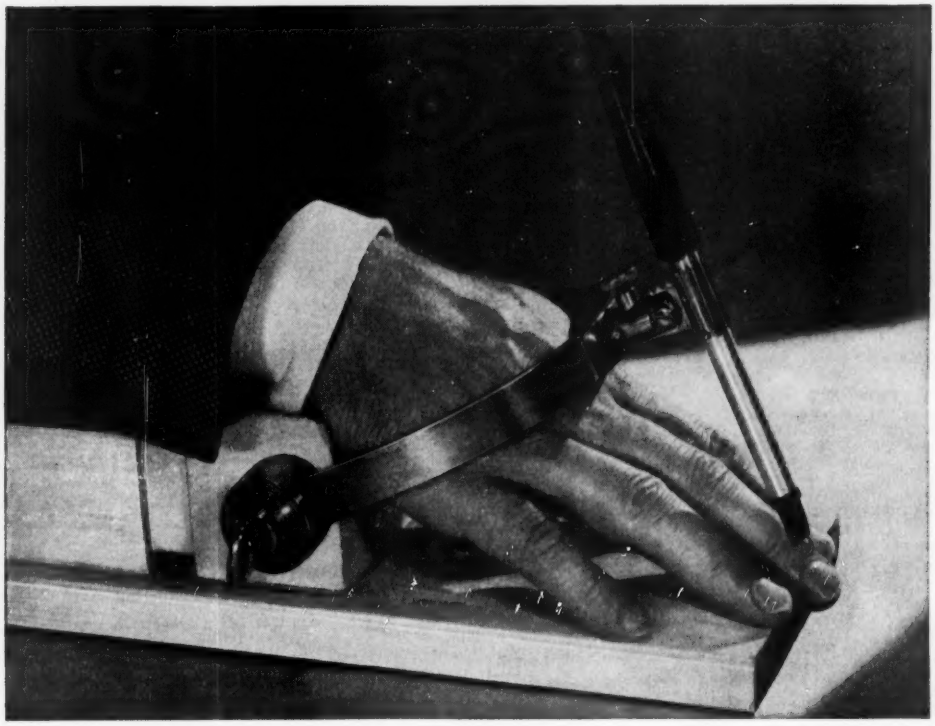

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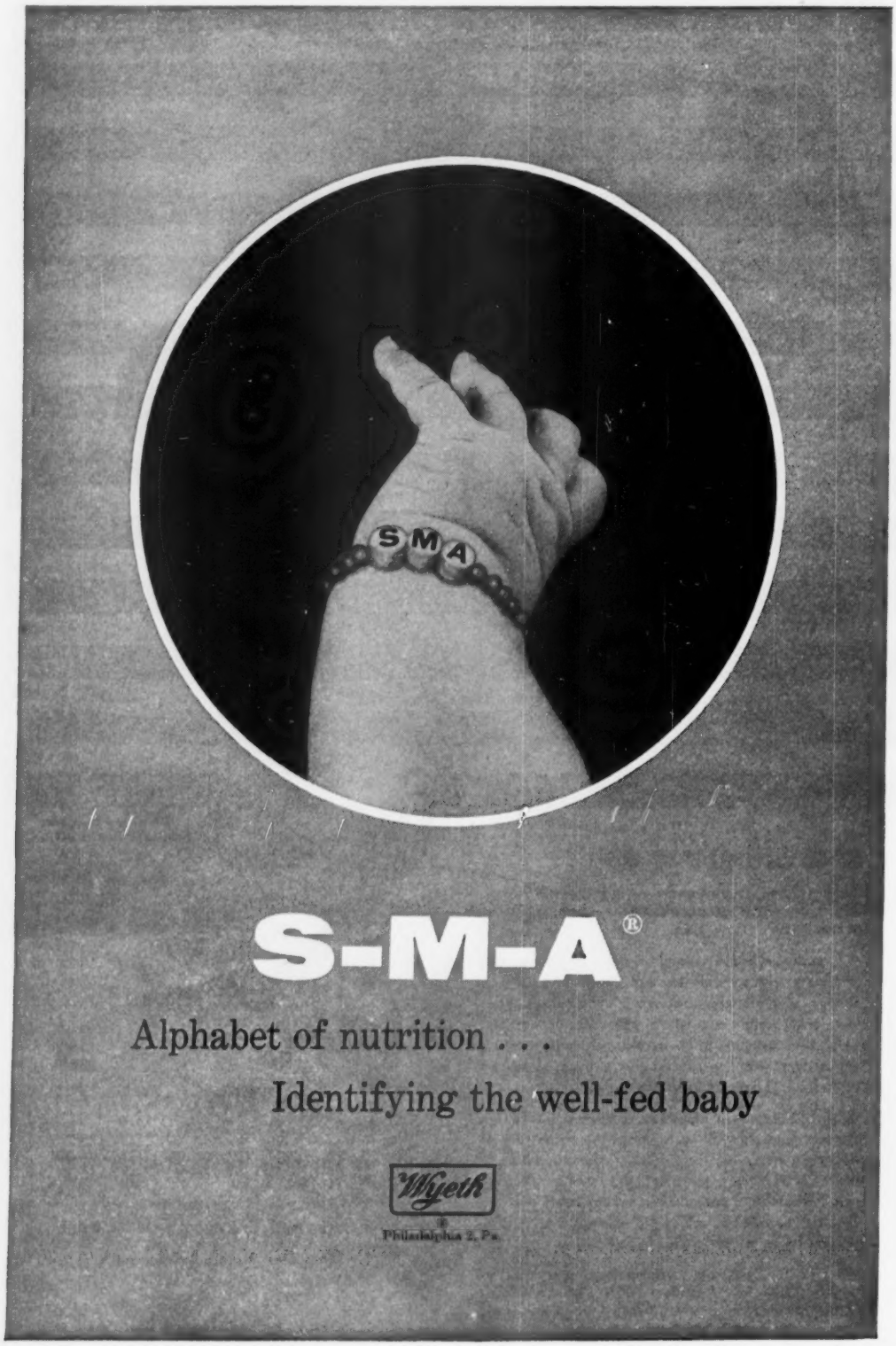
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Volume 251 AUGUST 19, 1954 Number 8

NEW PATHS TO A GROWING PROBLEM*

Contributions of Public Health to the Control of Chronic Disease

THOMAS PARRAN, M.D., DR. P.H.†

PITTSBURGH, PENNSYLVANIA

WHEN my long-time friend and colleague, John Gordon, asked me to give the Cutter Lecture this year, he explained that today he would be out of the country working on a project supported by the Rockefeller Foundation. He asked me to discuss the "existing position of public health in the approach to a control of chronic disease"; to "sum up what came out of the meetings themselves" at the Conference on the Care of the Long-Term Patient in Chicago in March, 1954; and to "give an assay of what needs to be done." It will be agreed that this was a formidable assignment.

No one of the three topics can be dealt with competently in a single lecture so that I have elected to deal with certain parts of each. In doing so, I am taking liberties that I would not venture if John Gordon were here instead of in India exploring what can be done about "the population problem."

Although my hosts may have been too modest to inform you, truth compels me to admit at the outset that there are several men on your faculty who are considered to be the leading American authorities on the subject of my discussion today: the ever-growing problem in this country of what can be done to prevent, to arrest, to cure, to alleviate, to rehabilitate and certainly to give better care to the millions of Americans who suffer from long-term chronic illness.

This problem has always been present. Among the several reasons why it is assuming greater importance today is the fact that in an aging population there are more chronically ill persons in proportion to the able-bodied; also, under the much more enlightened laws on care of the unfortunate, more care is being given, and it is costing more year by year. Perhaps, too, it is only now that all the health sciences from clinical medicine to hospital administration, nursing, social work and psychology — to mention a few of the many disciplines involved — have reached sufficient stature to engender hope of the effectiveness of their closely co-ordinated action in any solution or partial solution of the problem.

For the past five years the many apparently complex problems of the chronically ill have been the subject of intensive study by a national Commission on Chronic Illness, of which I am a member. The Commission was preceded and sponsored by a joint committee of representatives from the American Hospital Association, the American Medical Association, the American Public Health Association and the American Public Welfare Association, which in 1946 issued a statement under the title, "Planning for the Chronically Ill." The first and most important recommendation in this statement was that "the basic approach to chronic disease must be preventive." For the long pull, no one can quibble at this philosophy, which was spelled out in somewhat greater detail by the National Health Assembly in 1948:

> A basic change is required in society's traditional attitude toward chronic illness and its victims. The realization that much illness of this type can be prevented and that many persons heretofore regarded as inevitably disabled can be rehabilitated to lead useful, happy lives must become the starting point for any genuine program of chronic disease control.

PUBLIC HEALTH AS A SOCIAL SCIENCE

It should be recalled that originally public health was primarily an engineering science; later it became a medical science as well, and now it is becoming a social science also. My remarks today are based on this larger current concept.

At the beginning of my professional life the major public-health problems had to do with the control of acute communicable disease; the technics of sanitary engineering, epidemiology, immunization, quarantine and public-health nursing being utilized. Half a generation later this specialty aimed at translating the available scientific facts about health into terms of personal action in the family; community action in the county, town, city or state; and national ac-

*The Cutter Lecture on Preventive Medicine, delivered at Harvard School of Public Health, May 6, 1954.

†Dean, University of Pittsburgh Graduate School of Public Health.

tion when federal authority or funds were needed.

In other words, during the first period, which came to a close toward the end of World War I, things were done *for* people to improve their health. Once the backs of the worst epidemics had been broken sufficient confidence was established in public-health programs so that it was possible to guide citizens — with some measure of success — into the much more satisfying and productive venture of participation in doing things for themselves and thus improving their health.

In my opinion the problem now is to develop new methods. The citizen leaders have proved themselves in substantial degree. The leaders of the many scientific disciplines — including the social sciences — have proved that they can talk and plan together for health, once each has learned the language of the others, without much wrangling about professional precedence. If they prove that they can operate together and with citizens — who may need for this purpose to learn several languages or jargons — some pilot-plant demonstrations in several communities on a total health program might prove a new approach not only to the problems of the chronically ill but also to many other unanswered health questions. For only in a total health program — in contrast to the conventional health-department program — can one create the optimum environment for healthful living, make diagnoses early enough to assure possible prevention, provide treatment good enough to ameliorate what cannot be cured and, for the incurable, follow through with rehabilitation, whose potentials are just beginning to be discovered.

Before this or any comparable approach can succeed, however, much must be learned about the total problem of the chronically ill.

For example, in 1949, at the first meeting of the Commission on Chronic Illness, the difficulty of early diagnosis in most chronic diseases was emphasized, as was the unavailability of control methods. Relatively little has been learned since then, although many studies — including those on arthritis and rheumatism in Pittsburgh — grew out of the 1951 National Conference sponsored by the Commission and built around its general theme, "Preventive Aspects of Chronic Disease."

At the 1951 Conference a chronic disease was defined as comprising all impairments or deviations from the normal that have one or more of the following characteristics:

Are permanent.
Leave residual disability.
Are caused by nonreversible pathological alteration.
Require special training of the patient for rehabilitation.
May be expected to require a long period of supervision, observation, or care.

The very fact that the Conference needed to appoint a committee to work out definitions and to make them an important part of its final report is some indication of how loosely a good many physicians have thought and talked about chronic disease.

Even today precise definitions are lacking. Yet I need not remind this audience that few public-health programs succeed without precision in defining the objective and clear agreement on the means of accomplishing it.

The second National Conference sponsored by the Commission on Chronic Illness was held in March of this year at Chicago. The theme of the Conference was "The Care of the Long-Term Patient." Such patients, it was agreed, were those "suffering from chronic disease or impairment who require a continuous or prolonged period of care; that is, who are likely to need or who have received care for a continuous period of at least 30 days in a general hospital; or care for a continuous period of 3 months in another institution or at home." It was estimated that this involved about 5,300,000 persons substantially disabled by chronic disease or other impairment.

The following objectives were set up for the Conference:

Identify the requirements of the long-term patient in the various stages and severity of his illness.
Examine existing methods of providing care, explore new methods, and enunciate principles which should govern needed changes.
Suggest patterns for desirable relationships between services, facilities, and programs.
Recommend various ways to improve the financing of long-term care.
Establish direction and suggest next steps for local, state, and national programs for the care of the long-term patient.

The 1954 Conference emphasized the care of the "long-term patient" in his home, the relation of home-care programs to the general and to the special hospital, to the nursing home and to other institutions and the integration of services and facilities for long-term care. My section dealt with the kinds and types of *research* badly needed for substantial progress. Another section recommended the principles that should govern financing of research, demonstrations and care and suggested the principal sources of financial support.

It is needless to say that more long-term health care is needed for more people because fewer short-term deaths are occurring.

It should be remembered, however, that the horizons of the health sciences are expanding not only because of the continuous accretions to scientific knowledge but also because of the growth of social responsibility among a large majority of Americans. Both facts open larger areas of public-health enterprise. These wider horizons are being recognized even in medical education. Ward Darley says, "If medicine has become and is to be further developed as a function of society, society itself must be used as a classroom, laboratory, and clinic."

This is by no means a new idea to those who are

engaged in public-health teaching, research or practice. Quite the contrary. Nevertheless, its general acceptance in medical education will give the physician a deeper understanding of health and disease and will result in significant changes in the type of medical care available in the future. For it must be remembered that the treatment of acute illness generally represents a much simpler doctor-patient relation than the care of chronic illness. A psychiatrist tells me that the term "old crock" is used by physicians who are unable to cure their patients and subconsciously develop hostility toward them.

Setting this aside, since I claim no competence in psychiatry, it is obvious that the proper care of patients requiring long-term care involves the co-ordination of a variety of professional and other skills as well as of diverse physical facilities into a "therapeutic community." The whole situation is frequently complicated by serious financial, social, economic and psychologic implications not only for the patient but also for his family, and frequently for the community as well.

Highly interesting demonstrations are being carried out on the care of the long-term patient at home. These demonstrations take on particular significance when it is realized that of the 5,300,000 chronically ill persons (including both the bed ridden and the ambulatory) who are estimated to require care in this country, about 4,000,000, or almost 80 per cent, are not in an institution.

From the point of view of many who have studied this problem, the extension and adaptation of the more successful of such plans may provide the answer for a large proportion of patients in need of care. As Dr. Martin Cherkasky points out, there are two reasons. The negative one is the shortage of hospital beds, the high cost of providing them and the great difficulty of staffing them. The more valid reason is that for suitable patients, properly organized care in the home, properly rendered by skilled personnel, may be the best possible care. For the chronically sick patient, Dr. Cherkasky believes, the opportunity of obtaining good treatment within the shelter and security of the family at home can be an important factor in improvement.

There is, however, general agreement among the clinicians and hospital administrators who know the most about this subject that medical care is now geared to acute illness and must be completely revised before it can become effective in dealing with the chronically ill. As Dr. Anthony Rourke says, the needs of the long-term patient are more complicated. He may have a multiplicity of diseases. In addition to definitive medical care he may need services to provide for the emotional, spiritual, economic, social and rehabilitative aspects of his condition. His restoration to community usefulness may be rapid, slow, delayed or subject to repeated relapse. His condition is rarely static. Obviously, the care that he needs cannot be organized in every home. Sometimes, such care in the home defeats its own end by bringing the family to the breaking point.

Outpatient services offer a great but as yet unrealized opportunity to meet the needs of many of the chronically sick. Some of the better clinics organized to care for patients in all economic groups are at Duke University, at Chicago and at the Vincent Astor Clinic in New York City.

In smaller communities of 25,000 to 50,000 there is a practically untouched potential. An important consideration — and one that all agree has been shamefully overlooked — is that the outpatient clinics not only should meet the medical requirements of their patients but also should be conducted in such a way as to preserve the dignity of the patient as a person.

The private physician's responsibility in meeting the diversified needs of a chronic patient is to provide all services needed for optimum care. This means that the doctor must understand how much help other health workers can give as well as recognize his own limitations. In every area where studies have been made, the private physician coping with chronic illness in the middle and lower economic brackets — regardless of his personal good will or clinical proficiency — ran into the same series of obstacles in carrying out his responsibility. These included unavailability of necessary trained personnel; his unawareness of the availability of services besides his own and those of the visiting nurse; and his unwillingness to use available services because he was unfamiliar with their benefits, thought that they carried the stigma of charity or feared that some of his professional prerogatives might be assumed by others. In other cases community agencies providing needed services were over-restrictive and were frequently unable, because of their lack of communication with other agencies, to provide the auxiliary teamwork needed by the patient.

The over-all obstacle everywhere, however, is money. It is a truism that chronic illness and poverty tend to become synonymous, sooner or later. Too many patients in long-term illness scrape the bottom of the barrel for food, clothing and shelter — to say nothing of doctors' bills. Too many community agencies have not the funds to supply needed services to all chronic patients who need them. The sum of these factors suggests a major reorientation of community planning and action for the care of the chronically ill. In this, as in past programs, public-health agencies must be the spearhead.

It is generally agreed that the major elements involved in better control of chronic disease are laboratory and clinical studies that add to knowledge of causation, leading to better prevention and treatment (discussion of this aspect of the problem is outside the

scope of this paper); and development of sound community programs for the long-term care of patients. It is this aspect that I propose to discuss.

Community Programs

What realistic and useful contributions can be made by the acquisition of new and more extensive facts concerning long-term, disabling illness and by the placing of this knowledge in the hands of those responsible for planning, organizing, administering, evaluating and financing programs for patients with long-term illness?

All programs start in response to a recognized need and should have clear objectives, which should be balanced against available personnel, facilities and other resources. Since these are never static, objectives need to be shifted accordingly.

Continuous inquiry into what is being done, why it is being done and how well is an indispensable way of avoiding error. Much of this investigation should be made by the operating agency itself, and plans for studies of efficiency of operation should be built into the program at the outset; this is only good housekeeping. Other studies are more difficult and involve complicated methodologies as well as value judgments.

Furthermore, sociologic inquiry into the cause and effects of chronic illness and the results of treatment must parallel studies of illness from the point of view of medical needs and resources if one is to push back the frontiers of the unknown.

So far, efforts have barely opened the door to another area of sociologic research involving the health of the individual patient in relation to that of the family unit. What is good for the one may not be good for the other.

Medical educators and practitioners are rediscovering the concept of the whole man and the fact that he must be reassembled from his fragmented parts if his ill health (his dis-ease) is to be dealt with competently. Public-health educators and practitioners are rediscovering that the concept of the whole man must be related to the whole family unit in which complex factors interact during the course of the illness of any member. Moreover, health or the lack of it is influenced by the total environment in which the patient lives, works and plays and in which he struggles for success, security, happiness and health.

These are large questions, which cannot be answered today with any precision. I am convinced, however, that answers are necessary and feasible. Research is the tool that will open the way.

Large though these questions may be, public-health practitioners are constantly aware that in considering *research* in *long-term* illness they are dealing with a small sector of a larger problem, whose parameter is difficult to draw.

How can one study the tides of Massachusetts Bay as an isolated phenomenon unrelated to the larger planetary, gravitational and astronomic forces that produce them? The problem of public health is even more difficult than the study of tides since one is dealing neither with recurring nor with static phenomena, but with dynamic change and nonrecurring movement as science progresses and social concepts broaden.

In Chicago some weeks ago the Committee on Research of the Conference attempted to keep its task within the predefined limits, even at the risk of considering local tides. In the end it was agreed that useful generalizations *can* be drawn from such intensive study of limited phenomena. Two areas of research were concentrated on: size and characteristics of the problem of chronic illness; and effects of such illness upon the family and the community. Cutting across both areas was the problem of methodology.

Size and Characteristics of Problem

For use at the Conference Mr. G. St. J. Perrott and his colleagues in the Public Health Service assembled a "Source Book on Size and Characteristics of the Problem," which provided in compact form available statistical information, including many hitherto unpublished data, in 69 tables and a brief descriptive analysis of the statistics. Valuable though this information was, Perrott and his colleagues were the first to point out its shortcomings.

In fact, it was recognized that much vital information on virtually every aspect of the subject was lacking — whether it was the care of the patient at home or in an institution, the way that the activities of agencies should relate to one another or the set of complex problems involved in the financing of medical care.

The Conference committee that studied the problem of financing started with the premise that the American people no longer looked upon adequate care for the long-term patient as a privilege, but as a right that should be available to all.

As a base line for the committee's recommendations, it was stated that the extension of voluntary health insurance is the single most important instrument for financing better care of long-term patients who can afford to pay the required premiums. This should be extended to cover outpatient and home care, as well as that in nursing and convalescent homes.

Supplementary to voluntary health insurance are the benefits derived from private and philanthropic agencies, workmen's compensation, union welfare funds, industrial, agricultural and professional contributory funds and the like. When the total of these fails to meet the costs of needed care for the chronically ill there is no alternative to the conclusion that society as a whole, through taxation, must meet the deficit.

Private philanthropy has made substantial contributions, particularly in the training of health and

rehabilitation personnel. A number of business leaders at the Conference, however, expressed the view that there is much more to be done in this direction. The suggestion was advanced that philanthropic health agencies (particularly those that solicit funds from the public each year for a particular type of illness) could benefit by pooling part of their resources in a common effort to foster public education, preventive measures, expansion of diagnostic and rehabilitation services and especially the basic research so sorely needed in the solution of problems involving the chronically ill.

Here, for example, are some questions that emerged clearly from the Chicago Conference as having particular relevance:

(a) What are the conditions — medical, social, and economic — leading to the need for long-term care? Who suffers from these conditions and in what degree? How old are they? Where are they to be found?
(b) What services are needed to care adequately for these patients?
(c) What does adequate care really mean? For what purpose is home care given? To help restore the patient to health or to lessen the costs of institutional care?
(d) How should agencies best be organized to provide needed services?
(e) How can the efficiency of agency operation be measured?
(f) How does one measure the net contribution of any activity intended to help sick people?
(g) How much does care for long-term illness cost? Who pays it and how is it paid?

Gaps in knowledge of the current prevalence, duration and severity of chronic illness were recognized by all participants in the Conference. Yet the rationality of any action program must be based upon a *sound gauge* of needs. Estimates of the facilities, personnel and types of services that a community requires for prevention, treatment and rehabilitation of any chronic condition, or even for the chronic conditions in any segment of the population, must now be based in large part on limited information derived from earlier years or from some other community.

Moreover, the translation of morbidity data into needs for service and the translation of such needs into estimates of the personnel and facilities required to provide services for long-term illness are complex procedures. The data must be based, of course, on medical judgment regarding diagnosis, type and potential value of treatment and on assumptions regarding the best way to organize to do the job.

As Elinson says, it is not yet known exactly what information is necessary before reasonably good estimates of needed care can be made. Even the two closely parallel intensive studies of chronic disease in Hunterdon County and in Baltimore are not using identical information-gathering methods.

Perhaps the most important element to take into account, however, is the *purpose* of care. Reasonable purposes range widely, from prevention of imminent loss of life by emergency medical treatment to the development of happy and productive persons by means of long-term counseling and the provision of a favorable environment.

For convenience, the purposes of needed care may be considered as falling into two classes: conservative and rehabilitative.

Conservative care is intended to prevent any harmful changes — to conserve the present health status. This term can also be applied to preventive measures, as well as to secondary prevention designed to halt the progression of disease. Nursing homes and other domiciliary institutions usually provide no more than the minimum.

Rehabilitative care is intended to produce an observable ameliorative change. It may include improvement of a pathologic condition, as in halting a respiratory infection by means of antibiotics. An increase in self-sufficiency, such as increasing mobility through use of a wheel chair, is a proper goal of rehabilitative care, as is the improvement in economic self-sufficiency by training for a new job within the physical limitations of the patient.

Some kinds of care have both conservative and rehabilitational implications. The hospitalization of a psychotic patient, for example, may be merely conservative care for him. At the same time it may be rehabilitative for members of his family who are relieved of the burden of responsibility for him at home and freed to live their own lives more fully.

It is an important principle that definitions of terms and methods of measurement must be dictated by specific research objectives, but for any comparability among like programs uniformity of definitions and measurements is indispensable. In any one program definitions should be held constant.

A substantial amount of knowledge is, of course, being assembled by those who plan and administer health and welfare programs and by those who make surveys of health conditions in various parts of the United States. That knowledge could be more useful to others if the terms used were defined with clarity, precision and uniformity.

All Conference committees recognized the need for precise definitions. Nearly all the reports of preconference study groups start with a definition of what is meant by the topic discussed — home care, general hospital, outpatient department and the like.

The development of a glossary on this subject would aid in promoting uniformity of interpretation, in applying the results of experience, in measuring needs for care, in providing services, in evaluating the results of service and in estimating costs. If standardized definitions were widely used the knowledge derived from the experience in any one program could more readily be translated into plans for other programs.

My colleague at Pittsburgh, Dr. John McGibony, some years ago undertook to develop a glossary of terms used in connection with hospital and medical care, since he had found that even in different units

of the Public Health Service the same word frequently held different meanings.

In assaying what public health should do as its contribution to the eventual control of chronic illness, I have leaned heavily on Dr. Cecil G. Sheps, of the Beth Israel Hospital in Boston, who has done excellent work in assessing the effectiveness of programs in operation.

Up to now I have attempted to show something of the shape and form of this great objective in what I believe to be the third period of public-health advance in the United States. I have refrained, deliberately, from attempting to blueprint the exact status and detailed responsibilities of public-health disciplines in the total team that will be required to control chronic illness, for the sound reason that this is a task to be dealt with community by community, with as great a variety of approaches as there is a variety of towns, cities and states.

In each situation, however, the trained public-health group should be in control. Any program should be set up as follows:

It should *gauge* the need for one as against another program directed against one rather than another aspect of chronic disease.

It should *establish* realistic goals.

It should *determine* present and potential resources.

It should *balance* resources against objectives.

It should *build* into every program measures for the study of its efficiency.

It should *inquire* in advance, and at regular intervals thereafter, about the over-all effectiveness of each action program (this evaluation should be made by an outside, disinterested observer).

It should *question* all new proposals for action on the basis of sound principles drawn up by the Commission during five years of intensive study.

It should *evaluate* what comparable communities are doing.

It should *adapt* the successes of other communities to specific requirements of its own problem.

It should *relate* what is to be done to what is (or is not) being done now, as well as to over-all objectives.

It should *ensure* the participation of the professional and the community groups in the initial study, in the planning and in developing proposals for action (control of chronic disease must be a team job from its inception).

It should *decide* what should be done in all relevant circumstances.

It should *motivate* the community to act in the interest of its own health, of saving its tax dollar and of meeting its civic responsibility.

It should *experiment* under controlled conditions in a pilot plant; if results are firm, expansion is inevitable.

Finally, it should *report* to sponsors, community, and contemporaries a clinical history of the venture, what has been done well or badly and why.

Continuous inquiry into what is being done, why it is being done and how well is an indispensable method of avoiding error. Research and action are inseparable. Since any action program is based substantially on research, to research it must return to renew its strength. This requires a continuous working relation between research people — the people who give service — and the people who pay for it.

Effects on Family and Community

One must face the fact that hundreds of thousands of families every year will be shattered and brought to the point of destitution by prolonged illness. In 1952, 500,000 families spent between 50 and 100 per cent of the total family income on medical care; another 500,000 families had medical expenses exceeding the total family income. Unless this burden of expense can be shared — or lightened through research — this vast tragedy will increase with the years.

Yet, at this moment, there is still not sufficient knowledge about the cost of chronic illness per se to assess surely what should be done as a nation, or what part states, cities and voluntary effort should contribute. This ignorance is a formidable barrier, not only in assessing needs, but also in assessing the efficiency of current services and evaluating how well they meet their stated objectives. In other words, to use industrial terms, one scarcely knows how good the product is or whether a different product could do the job better.

It seems unnecessary, in an industrial society whose phenomenal efficiency and constantly expanding productivity are founded upon research, to argue at any length about the need for comparable research on the problems of chronic illness.

The national investment in laboratory and clinical research related to medicine is $200,000,000 a year. It seems reasonable to suggest that an additional investment be made to translate the knowledge gained into action for health. To do this more precise information regarding the health needs of the nation and the best ways of meeting them is essential.

An impressive brief could be drawn up for the economic necessity of controlling chronic disease. Even with the great ignorance about the details of the problem and uncertainty about method, except through fractional approaches, one knows approximately the millions of persons involved and can estimate the appalling cost of the utterly inadequate care the majority receive.

But when statistics are discussed the problem becomes remote. The suffering becomes impersonal. Always behind the figures are the human beings, the broken lives. Like any action to help those less fortunate, the vast program now in the shaping to improve the care (and thereby shorten the long help-

lessness) of the chronically ill can succeed only if it is motivated by conscience and compassion and soundly based on knowledge that is the fruit of research.

Conclusion

Science is the rock on which one must build, but the student of public health will not forget that science alone is not enough. He need only be reminded — if I may paraphrase Sir Arthur Newsholme — that twinges of conscience have proved the most powerful antiseptic, and that the sentiment against suffering is the greatest healing force of the present time.

WEDGE RESECTION FOR POLYCYSTIC OVARIES*

A Critical Analysis of 40 Operations

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In 1935 Stein and Leventhal[1] reported 7 cases of amenorrhea associated with polycystic ovaries. In addition 4 of their patients complained of hirsutism, and 3 were obese. After wedge resection of the ovaries, normal menses reappeared in all 7 patients, 5 of whom eventually conceived. The amenorrhea and the polycystic ovaries were thought to be due to an imbalance of the pituitary-ovarian axis. The combination has become known as the Stein–Leventhal syndrome.

Secondary amenorrhea as the main clinical symptom of this syndrome was emphasized by Leventhal and Cohen[2] in 1951, when they stated that wedge resection was not indicated unless this menstrual abnormality was present.

That menometrorrhagia associated with polycystic ovaries can be corrected by wedge resection, however, was shown by Robinson[3] as early as 1935. As more reports appeared in the literature it became readily apparent that polycystic ovaries could be found in association with any type of menstrual irregularity,[4-7] having even been reported in patients who bled regularly from a secretory endometrium.[8] Furthermore, it was shown that polycystic ovaries were present in numerous cases in which the menstrual irregularities were associated with an extragenital endocrine abnormality such as Cushing's or adrenogenital disease. The literature on this subject was adequately reviewed up to 1951 by Du Toit.[9]

Since a certain amount of disagreement and possible confusion has resulted from the various interpretations of this syndrome it was decided to attempt to clarify some aspects of it by a critical review of the patients operated on at the Sloane Hospital for Women. Two basic questions for which an answer was sought were whether polycystic ovaries occurred with all sorts of menstrual disturbances in addition to amenorrhea and whether they constituted a coincidental finding in cases of extragenital endocrine disease such as adrenogenital syndromes. Furthermore, if the answer was in the affirmative, for which type of case was the operation of wedge resection most successful?

Even when wedge resection was restricted to cases that fulfilled the criteria of Leventhal and Cohen[2] all follow-up reports showed a number of cases in which the operation failed to correct the menstrual abnormalities.[8-11] It is tempting to speculate that, in at least some of these patients, the menstrual irregularities and polycystic ovaries were secondary to extragenital disease that had been overlooked. Obviously, in these patients, wedge resection would be of no avail. Exhaustive endocrine studies were not done in most of the published cases, and without these studies Cushing's or adrenogenital disease that was not severe may have remained undiagnosed. The combination of amenorrhea, hirsutism and obesity is a feature of many endocrine syndromes. On the other hand it is imperative to define which syndrome is amenable to wedge resection and which is not.

Forty cases of polycystic ovaries in which operation had been performed were collected from the files of the Sloane Hospital for Women. Regardless of preoperative symptoms, all patients who at laparotomy were found to have polycystic ovaries were included in this study. It was obvious that this was a highly selected group. Most patients were admitted through one of the gynecologic clinics, and the incidence of patients with predominantly gynecologic symptoms was therefore higher than that in a nonselected group. Some patients had been seen first by other departments, but only when disease other than polycystic ovaries had been excluded were they referred to the Department of Gynecology.

It was thought, however, that even though the cases were selected in this manner, the findings were of sufficient clinical importance to aid somewhat in the clarification of this entity.

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Clinical Data

Menstrual Cycle

Table 1 summarizes the pattern of menstruation. Considerable overlapping of categories was present. In some patients episodes of amenorrhea were occasionally interrupted by long periods of metrorrhagia and in others amenorrhea alternated with oligomenorrhea. In patients with irregular menses bleeding from anovulatory endometrium was the rule. That ovulation can occur even after long-standing amenorrhea, however, is shown by the fact that 1 patient with polycystic ovaries, identified at culdoscopic examination, who refused wedge resection conceived after amenorrhea for sixteen months. Furthermore, in 2 patients a normal corpus luteum was found in the resected ovaries.

Table 1. *Menstrual Cycles in 40 Patients (Relation of Other Complaints to Menstrual Disturbances).*

Menstrual Pattern	No. of Patients	Patients with Hirsutism	Obese Patients
Amenorrhea (more than 6 mo.)	14	13	8
Oligomenorrhea (less than 6 mo.)	16	12	7
Totals	30	25	15
Regular cycle	3	2	0
Menometrorrhagia	7	0	0
Totals	40	27	15

Thirteen patients had one or more endometrial biopsies. In patients with amenorrhea atrophic, inactive proliferative or no endometrium was obtained. In 1 case of oligomenorrhea and in 2 of menometrorrhagia hyperplastic endometrium was found. No case with secretory endometrium was noted.

In several patients endometrial response was tested with estrogens. Normal proliferation of the endometrium, followed by withdrawal bleeding, was invariably seen.

Hirsutism

Hirsutism was found in 27 patients, an incidence higher than that usually reported, probably because of the technic of selection. Correlation of hirsutism with the type of menstrual cycle is shown in Table 1. The difference between the incidence of hirsutism in the group with oligomenorrhea and amenorrhea and in the group with menomenorrhagia is striking. In 11 patients some enlargement of the clitoris was present. Acne occurred in a variable degree in 15 patients. Three complained that their voices had become lower. None had temporal hair recession or atrophy of the breasts. No uniform pattern of hirsutism was present, nor was there correlation between the degree of the hirsutism and the presence of additional symptoms of virilism.

Sterility

Sterility was an additional complaint in 14 cases, in 2 of which it was secondary.

Endocrine Investigation

In an attempt to ascertain whether or not extragenital endocrine disease could be detected some of our patients were subjected to an exhaustive investigation. For the reasons of selection discussed above, the chances of this kind of abnormality in our patients should have been less than those in a true cross section of patients with polycystic ovaries.

In no case could hypothyroidism or hyperthyroidism be proved. The basal metabolic rate was determined in 25 patients, and all values were within normal limits. In 6 uptake of radioactive iodine and cholesterol determinations were done as additional tests; again, all values were normal except that in 1 patient the uptake of radioactive iodine was low although she was euthyroid in all other respects. No recent medication could account for this abnormal value. Thyroid therapy in a number of patients had been completely unsuccessful.

In view of the high incidence of hirsutism and other symptoms of virilism in patients with polycystic ovaries, as well as the frequent reports in the literature of polycystic ovaries in association with Cushing's and adrenogenital disease, special attention was given to adrenal function.

Attempts to correlate the frequent finding of hirsutism in patients with high levels of "androgens" have been generally unsuccessful.[2,4,5,6,9,12,13] In most cases reported in the literature normal values of urinary 17-ketosteroids have been found. High levels in some cases were mentioned by Plate,[6] Rottinghuis,[7] Du Toit[9] and Dorfman et al.[14] This lack of association between hirsutism and the levels of 17-ketosteroids is not surprising. Many pathways are known through which "androgens" can be metabolized without causing significant rises in the urinary 17-ketosteroids. In such extreme cases of masculinization as those associated with arrhenoblastomas and other virilizing ovarian tumors normal 17-ketosteroids are often found. This rise, however, is almost invariably present in hirsutism and virilism of adrenal origin.

Total 17-ketosteroids were tested in 26 cases. In our laboratory, where the modified method of Holtorf and Koch is used, normal values range between 5 and 15 mg. per twenty-four hours. In 23 cases the values were within normal limits. There was no difference in the total 17-ketosteroids excretion between patients with and those without hirsutism. In 3 cases, however, values were repeatedly above the normal limits. Brief abstracts of these cases are as follows:

Case 1. J. S., a 22-year-old woman whose menarche began at the age of 12, at first had irregular menses but had had regular cycles for 1 year. Increased hair growth began at the age of 16, with no obesity; 17-ketosteroids were between 17 and 22 mg. per 24 hours. At laparotomy bilateral

polycystic ovaries and full-blown corpus luteum were found, and wedge resection was done. On follow-up examination the menses were regular, but the hirsutism was unchanged; pregnancy occurred 1 year later.

CASE 2. In A. T., a 20-year-old girl, normal menstruation began at the age of 14, lasting till age 18, when periods became irregular, with episodes of amenorrhea lasting as long as 6 months. Hirsutism was first noticed at the age of 15, but a sudden increase occurred at 18. The clitoris was normal, and no other signs of virilism were noted. The patient also complained of acne. The serum cholesterol, basal metabolic rate, x-ray films of the skull and adrenal aerograms were normal; 17-ketosteroids were 19.2 mg. per 24 hours or over on several occasions.

Wedge resection was done in 1944. A follow-up study 10 years later showed the hirsutism to be unchanged, the 17-ketosteroid excretion still above normal and menstruation occurring irregularly about every 6 or 7 weeks. Since the patient was single an endometrial biopsy was not obtained, nor did the problem of sterility present itself.

CASE 3. In H. F., a 30-year-old woman, the menarche had occurred at 11 years of age and was regular till the age of 18. At this time the patient skipped a period and since then had had periods every 3 or 4 months. Hirsutism was noticed at the age of 20.

When seen in 1952 she had had amenorrhea for 1 year, with extensive hirsutism. At laparotomy polycystic ovaries were found and resected. Bleeding appeared 1 month later and was anovulatory. At this time further investigation revealed a normal sella turcica, but slight osteoporosis on x-ray study. The blood pressure was 150/90, but glucose tolerance was normal and, although fairly obese, the patient had no striae.

Three 17-ketosteroid determinations were 22, 26 and 24 mg. per 24 hours,—a consistently high result,—and the 11-oxysteroid determinations were also slightly elevated. An adrenal aerogram was normal. Although incipient Cushing's disease was considered, it was thought that not enough data were present for us to make this diagnosis (the slight elevation in 11-oxysteroids is often seen in patients with high 17-ketosteroids and was probably not significant).

The next period began 7 weeks later and was again anovulatory. Cortisone was administered for reasons described below, and the level of 17-ketosteroids dropped to 7 and 8 mg. per 24 hours. During the 2 months on this therapy the patient had 2 ovulatory menses at 4-week intervals. On withdrawal of therapy anovulation reappeared, and the interval between the periods increased.

Although these patients had enlarged, polycystic ovaries follow-up studies indicated that wedge resection was of no particular help to them. To be sure, Case 1 became pregnant a year after operation, but she actually had a corpus luteum present at the time of operation and so was ovulating and presumably could have become pregnant before operation occurred. The evidence deduced from these 3 patients confirms the impression that consistently high 17-ketosteroids levels should lead one to suspect the adrenal gland rather than the ovary as the offending organ even though the ovary is polycystic.

It is well known that the value given for total 17-ketosteroids is a composite one, being the sum of the concentrations of the different 17-ketosteroids present in the urine. Some of these 17-ketosteroids are neither androgens nor metabolites of androgens.

It has been speculated that in the so-called Stein–Leventhal syndrome, although the value of the total 17-ketosteroids remains within normal limits, the "androgenic fraction" of the complex may be elevated or abnormal 17-ketosteroids may be present. Separation of the different steroids present in the 17-ketosteroid-complex is possible by chromatographic technics. Using column chromatography, Holmer,[12] Plate,[8] Du Toit[9] and Rottinghuis[7] could not find a consistent pattern in patients with polycystic ovaries. However, the technic of these authors did not actually identify these steroids, but merely postulated their presence by their position in the chromatogram.

By means of column chromatography, with subsequent identification of the isolated steroids by infrared analysis, fractionation of the 17-ketosteroid complex was done in 7 patients with polycystic ovaries. Normal values were found in all but 1, who showed an abnormally high percentage of dehydroisoandrosterone. Increased excretion of this steroid is considered to be a sign of adrenal hyperactivity. In 3 patients chromatograms were taken before and after a successful wedge resection, and no change in the chromatograms was observed.

Cortisone Test

Wilkins et al.,[15] Bartter et al.[16] and Jailer et al.[17] have demonstrated that the administration of cortisone to patients with adrenal hyperplasia results in a fall of the urinary 17-ketosteroids from a characteristically high level to values that are approximately normal for the sex and age of the patient. In the postpubertal female this is accompanied by the onset of a normal menstrual cycle. This decrease in 17-ketosteroids is due to the inhibitory effect of cortisone on ACTH production by the pituitary gland, resulting in "a hormonal adrenalectomy" and the disappearance of the abnormal adrenal secretion by lack of ACTH stimulation. A similar drop of the elevated 17-ketosteroids to normal levels on cortisone therapy has been shown by Jones et al.,[18] Greenblatt[19] and Van de Wiele, Jailer and Gold[20] in patients with "hirsutism" associated with elevated 17-ketosteroids. In these patients with irregular menses a normal menstrual cycle reappeared. It seems logical to assume that if the amenorrhea and the polycystic ovaries in the Stein–Leventhal syndrome are secondary to adrenal hyperactivity, a similar response to cortisone therapy should be seen. Therefore, 50 mg. of cortisone a day for thirty days was given to 3 patients with polycystic ovaries; all 3 were hirsute and had persistent anovulation as proved by repeated endometrial biopsies. Steroidal studies, including total 17-ketosteroids, chromatographic separation of the 17-ketosteroids, pregnanediol and total corticoids, were done before medication and found to be negative. On this therapy no change in the clinical picture was observed. The basal-body-temperature chart remained flat, and endometrial biopsies showed no change in comparison with the picture before treatment. It was therefore assumed that the symptoms were not due to adrenal pathology. All 3 were subjected to wedge resection, and normal ovulatory menses occurred postoperatively.

Pituitary Gland

It has been postulated by some authors[1,5,6,12] that hyperactivity of the pituitary gland is a cause of polycystic ovaries. Injection of chorionic gonadotrophins

in guinea pigs produces ovarian changes similar to those seen in the human polycystic ovary.[21] This is accompanied by unmistakable signs of virilization, an effect that is mediated through the ovary since it disappears when the animal has been castrated before the injection. However, it should be stressed that these ovarian changes, as well as the virilization, can be produced by transplantation of the ovaries,[22] subtotal castration[23] and irradiation of the ovaries,[24] as well as by injection of chorionic gonadotrophins.

Determinations of follicle-stimulating hormone in patients with polycystic ovaries have been done by Ingersoll and McDermott,[6] Plate,[5] Du Toit[9] and Rottinghuis.[7] Although normal values were found in the majority of patients, in some the hormone was not present in measurable amounts. Six of our patients were tested for excessive amounts, and values were normal. No satisfactory method for measurement of luteinizing hormone has been developed, so that tests for this gonadotrophin could not be done.

X-ray examination of the skull in 22 patients failed to show any abnormalities of the sella turcica. In 2 patients, however, hyperostosis frontalis was present. We do not know the significance of this finding.

Results

Wedge resection was done on 40 patients, 27 of whom were hirsute.

Influence on Hirsutism

Follow-up reports for this symptom were obtained in 25 of the 27 patients who were hirsute. In only 1 did the abnormal hair distribution revert to a normal pattern. In 4 the rate of hair growth increased after operation. In the remainder the hair distribution was static.

Changes in Menstrual Cycle

The effect of wedge resection on the menstrual cycle could be evaluated in only 28 patients. The others were excluded either because they had normal menses before wedge resection or because the follow-up period was too short or incomplete. The following postoperative results apply to patients who had irregular menses preoperatively:

Four patients conceived soon after the wedge resection. However, only 14 cases included in this study were sterility problems. In addition, in 2 of these, male-sterility factors were present.

Sixteen patients had normal cycles postoperatively as determined by basal body temperature or premenstrual biopsy.

Four patients had normal cycles after operation, but on repeated biopsies *occasional* anovulation was seen.

In 4 patients wedge resection completely failed to correct the menstrual irregularities. Three of these had amenorrhea preoperatively. The fourth had menometrorrhagia.

It can be said, therefore, that of 28 patients, 20 were cured, 4 improved and 4 unaffected.

Discussion

From the data presented we believe that a number of conclusions are warranted. It is apparent that the syndrome under discussion, presenting symptoms of menstrual abnormality, usually amenorrhea, frequent hirsutism, usually with obesity and the physical finding of enlarged polycystic ovaries, is probably due to some complex and as yet not understood dysfunction of the pituitary-adrenal-ovarian axis. No type of therapy seems to result in a disappearance of the hirsutism — at least not for long. However, the menstrual disturbances, anovulation and sterility are cured by wedge resection in a high percentage of cases in which the ovary seems to be the main offender whereas it is a failure when the main source of physiologic misbehavior is apparently the adrenal gland.

In answer to the questions posed in the first part of this paper, then, a review of these 40 cases discloses that any type of menstrual disturbance may occur in the presence of polycystic ovaries (but usually the menstrual symptom is amenorrhea) and that polycystic ovaries are present even when the predominantly abnormal function is of the adrenal gland.

What criteria are available, then, for determining whether or not wedge resection will be of value or of determining which case is predominantly ovarian and which is predominantly adrenal in etiology?

Although the determination of specific 17-ketosteroid excretion is still technically difficult and is being subjected to further critical investigation the finding of consistently high excretion levels of these steroids in these patients immediately places suspicion on the adrenal gland as the offending organ in the complex linkage. Confirmatory evidence of this impression is noted if endometrial biopsy and basal body temperature indicate occasional ovulation in patients with infrequent periods.

What has proved to be a further valuable diagnostic aid is the "cortisone test." When adrenal depression is produced by cortisone and the amenorrheic patient resumes menstruation and ovulation during the period of administration of this hormone, it seems reasonable to assume that abnormal adrenal physiology is playing a predominant role in the patient's difficulty.

Conclusions

When 17-ketosteroids are within normal limits, when the patient is consistently anovulatory (even though she may be bleeding irregularly) and when adrenal depression with cortisone does not result in ovulation and regular menstruation wedge resection

will probably cure the patient's menstrual abnormalities and sterility.

In patients whose chief complaint is hirsutism little hope can be given that wedge resection will improve this symptom. However, it may be worth while to perform the operation at the first sign of hirsutism when it is indicated, in the hope of arresting hair growth.

It is our impression that the Stein–Leventhal syndrome is a symptom complex whose true etiology is not as yet known but that the unknown motivating dysfunction may affect either the adrenal gland or the ovary or both. It is also thought that, if cases are appropriately selected, cure of menstrual dysfunction but not of hirsutism can be effected in a high percentage of patients.

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MITRAL STENOSIS IN FACSIMILE*

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BALTIMORE

SINCE surgical treatment for mitral stenosis has become an accepted procedure it behooves the clinician to exert the utmost diligence in selecting all candidates who could conceivably be benefited by commissurotomy. With the increased interest in recent years, it has been more widely recognized that the "classic signs"—that is, apical mid-diastolic and presystolic murmur, accentuated mitral first sound, loud and reduplicated pulmonic second sound and mitral "opening snap"—are not always present in mitral stenosis, and means of ascertaining with certainty the presence or absence of physiologically significant obstruction are being sought. Cardiac catheterization, angiocardiography, roentgenokymography and electrokymography, and other means of cardiovascular study have greatly increased understanding of certain phenomena of physiologic importance, but cases still exist in which the presence or absence of mitral stenosis remains in doubt. Experience in recent months with several patients who were thought to have mitral stenosis but at operation or autopsy proved to have other lesions leads us to report the following cases.

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Case Reports

Case 1. E. R. (J.H.H. 647000), a 44-year-old housewife, was admitted to the hospital on July 27, 1953, because of intractable ascites. The family history was significant in that a sister was described as having "swelling" similar to that of the patient. The past history was noncontributory, except as described below. There was no history of rheumatic fever.

Fifteen years previously the patient's only living child was delivered, after which she had persistent weakness. Seven or eight years later she began to have a "choking sensation," with nervousness. She experienced substernal pain with exertion on one occasion four and a half years before admission and, 3 weeks after this, cough, fever and pain in the right anterior aspect of the chest diagnosed as pneumonia and treated with penicillin. After approximately 1 week edema of the legs followed by abdominal swelling developed. The cough, which was nonproductive, persisted for 3 years and then disappeared spontaneously. The edema and abdominal swelling gradually increased, and for this reason therapy with mercurial diuretics was begun. Initially, these elicited diuresis, but refractoriness developed. The swelling did not totally clear with this regimen. From the onset orthopnea persisted. Beginning 2 years before admission and continuing until admission, frequent abdominal paracentesis had been necessary. Various therapeutic efforts, including full doses of digitalis, were undertaken, always without benefit. A liver biopsy, performed elsewhere during the year before admission, was said to have shown "early portal cirrhosis."

Physical examination showed an emaciated, chronically ill woman in no acute distress. The skin was pale and dry, and there was a scaling, erythematous eruption over both lower legs. The mucous membranes were pale. There was marked distention of the neck veins. Both leaves of the diaphragm were high, and there were scattered dry rales in the right-lower-lung field posteriorly. The heart was enlarged in all diameters, with the borders of dullness extending to the anterior axillary line in the 5th interspace on the left and 3 cm. lateral to the right sternal border in the 2d and 3d spaces on the right. There was a prominent precordial bulge. No thrill was palpable. The mitral 1st sound was not accentuated, although apical sounds were distant (after diuresis the mitral 1st sound became distinctly louder than it had been on admission). At the apex there was a faint, blowing systolic murmur and a rumbling, mid-diastolic murmur heard best with the patient in the left lateral decubitus or upright position. (This murmur was noted to disappear at times, although it did not appear to be influenced by position.) There was a protodiastolic gallop. The pulmonic 2d sound was loud and split. The abdomen was distended with ascites. The liver edge, which extended 5 fingerbreadths below the right costal margin, was firm and nontender. The tip of the spleen was palpable. There was a metal Cooney button in the right lower quadrant subcutaneously and a large ventral hernia. There was pitting edema extending to the midlumbar portion of the spine. There was no cyanosis or clubbing.

The temperature was 97.6°F., the pulse 88, and the respirations 20. The blood pressure was 116/78.

Examination of the blood revealed a hematocrit of 39 per cent, with a corrected sedimentation rate of 7 mm. in 1 hour (Wintrobe method), and a white-cell count of 5000, with a normal differential. Serologic tests for syphilis were negative. Urinalysis demonstrated a trace of albumin but was otherwise normal. On admission the nonprotein nitrogen was 37 mg., and the protein 6.3 gm. per 100 cc., with 4.0 gm. of albumin and 2.3 gm. of globulin. The carbon dioxide was 31.8 milliequiv., chloride 93.4 milliequiv., and sodium 125 milliequiv., and potassium 4.5 milliequiv. per liter. Liver-function tests were negative except for an alkaline phosphatase of 12.5 Bodansky units.

Venous pressure in the left antecubital fossa was equivalent to 310 mm. of glucose. An electrocardiogram showed right-axis deviation, with strain of the right ventricle. A ballistocardiogram was grossly abnormal. X-ray study of the chest disclosed considerable cardiomegaly, including the pulmonary artery, left atrium and both ventricles. There were bilateral pleural effusions. Fluoroscopic and roentgenokymographic examinations revealed limited pulsation of the heart borders.

Cardiac catheterization was performed by Dr. Henry T. Bahnson. The pressure in the main pulmonary artery was 91/34 mm. of mercury, with a similar elevation of pressure in the right ventricle. The pressure in the right atrium was 16/8 mm. of mercury. There was no evidence of intracardiac shunt. The cardiac index was 2.8 liters.

On admission the patient was kept at bed rest and on a high-calorie, low-salt diet. She was digitalized with leaf digitalis, and maximal diuresis with ammonium chloride and mercaptomerin was accomplished. An abdominal paracentesis was performed, with removal of 6600 cc. of transudate. The weight fell a total of 6 pounds with diuresis after the paracentesis, but ascites and edema persisted. After what was considered maximal preparation an exploratory operation was performed by Dr. Bahnson under thiopental and ether anesthesia. (The preoperative diagnosis was "probable mitral stenosis.") The left atrium and left pulmonary vein did not appear to be enlarged. The operator's finger was inserted into the atrium, and a golf-ball-sized tumor could be felt, attached to the interatrial septum. The pedicle seemed small, and with evaluative manipulation the tumor was dislodged from its base. An attempt was made to deliver the tumor into the pulmonary vein, but it escaped, and when efforts were made to trap it in the ventricle it was fractured, with a large portion embolizing into the aorta, and a smaller portion recovered. The heart action weakened and, in spite of stimulants and use of the electric defibrillator, could not be restored to normal. The portion of the tumor removed at operation had the gross appearance of myxoma, microscopical sections of which showed well encapsulated, loose, myxomatous stroma, in which there were small and large capillary spaces.

Autopsy revealed a heart weighing 390 gm. The right atrium and ventricle were hypertrophied and dilated, and the left ventricle atrophic. The valves were normal. There was an ovoid patch of roughening on the wall of the left atrial septum (1.5 by 2.0 cm.) that may have represented the site of attachment of the myxoma, but no remaining tumor could be identified (a large fragment of myxomatous tissue had lodged as an embolus in the arch of the aorta). Gross and microscopical study of the remaining organs showed chronic passive congestion, and there was severe pulmonary arteriosclerosis.

This patient presented evidence of obstruction of flow into the left ventricle, with resultant congestion of the lesser circulation, and right-sided heart failure. Physical findings were compatible with mitral stenosis, but the absence of a history of rheumatic fever, the changing murmur and the failure to respond adequately to digitalis should have led to suspicion of intracardiac tumor. At operation the myxoma was completely removed from the left atrium, and if the fragments had been successfully delivered and embolism prevented, a surgical cure could have resulted.

Case 2. L. P. (J.H.H. 609628), a 19-year-old girl, was referred to the hospital for evaluation for cardiac surgery in June, 1952. The family and past histories were noncontributory. There was no history of rheumatic fever. The present illness had apparently begun when a heart murmur was first discovered on routine examination at the age of 10. The patient believed that she had had mild exertional dyspnea all her life. Three months before admission she experienced a small hemoptysis; 2 months later she noted recurrent small hemoptyses over a 4-day period.

Physical examination disclosed a well developed and well nourished girl in no acute distress. There was no clubbing or cyanosis. The lung fields were clear. The heart was enlarged, with the point of maximal impulse 1.5 cm. lateral to the midclavicular line in the 5th interspace. No thrill was felt. The mitral 1st sound was loud, and the pulmonic 2d sound accentuated and split. At the apex there was a clear, low-pitched, mid-diastolic murmur. Physical examination was otherwise unrevealing.

The temperature was 99°F., the pulse 96, and the respirations 16. The blood pressure was 95/60.

Routine laboratory studies of cytologic and chemical constituents of the blood were normal. Urinalysis showed no abnormality. An electrocardiogram revealed changes compatible with strain of the right and left ventricles, with broad, notched P waves. Radiographic and fluoroscopic examinations of the heart demonstrated considerable enlargement of the left atrium, with enlargement as well of the pulmonary artery and right side of the heart. These were interpreted by the fluoroscopist as compatible with disease of the mitral valve (and congestive changes in the lungs).

Cardiac catheterization was performed by Dr. Bahnson. Pressure in the pulmonary artery was 48/22 mm. of mercury. The cardiac index was 2.12 liters. No evidence of a shunt was identified. Pulmonary vascular resistance on breathing of room air was 9.45 mm. of mercury per liter per square meter, and on breathing of 40 per cent oxygen was 8.67 mm.

After completion of these studies the patient was discharged to return for commissurotomy. It was the clinical impression of all observers that she probably had mitral stenosis. She returned in October, without having experienced any additional symptoms. Examination revealed only 2 changes of note: a late systolic apical "scratch" and fine bilateral basilar rales.

On October 6 anterior thoracotomy was performed on the left by Dr. Alfred Blalock under thiopental, curare and ether anesthesia. After the left atrium was entered he observed that the mitral valve differed from that in the usual

case of mitral stenosis and made the following operative note:

> There was no evidence of mitral regurgitation. I had difficulty in locating the mitral orifice. In the first place the left atrium was tremendous; with my finger introduced to the fullest extent I could not feel the septum. I finally located a small mitral orifice. It was located immediately adjacent to the lateral part of the left-ventricle wall and was somewhat further posterior than usual. I got the impression that the aortic mitral leaflet was quite large and that the posterior leaflet was quite small. The valve cusps were so soft that it was difficult to tell exactly what I was palpating. At any rate, I could feel a small orifice, which was about 8 mm. in diameter. This was dilated, and the posteromedial commissure was torn. This was accomplished very easily, for there was little thickening of the structures. I also dilated and I think partially tore the anterolateral commissure. At any rate the orifice was increased considerably, to probably 4 or 5 times its original size. I had the impression that the inflow tract was smaller than normal. When my index finger was thrust down into the ventricle it seemed to occupy the greater part of the cavity.
>
> I think this valve was different from any on which we have operated, and I wonder if this lesion might not be a congenital one. There was certainly very little thickening of the valve cusps, and I have not had a previous case in which it was so difficult to find the mitral orifice.

The postoperative course was complicated by the appearance of gradually increasing heart failure. By the 5th day the venous pressure had risen to 138 mm. of water, and the circulation time to 40 seconds. Increasing basilar rales and, later, bilateral pleural effusion developed. The patient was placed on sodium restriction and diuretics (digitalization had been accomplished before operation), and thoracenteses were performed. Her condition seemed to improve temporarily, but she died suddenly on the 12th postoperative day.

The final clinical impression, in view of the operative findings, was obstruction to the outflow and inflow tracts of the left ventricle with possible congenital mitral stenosis.

Autopsy revealed a heart weighing 410 gm. Both atria were enlarged; the right ventricle was dilated and hypertrophied. The tricuspid ring was dilated, and the pulmonary artery thickened. The mitral ring and valve were delicate. There was a tear in the septal wall on the left at the site of the anterior papillary muscle. There was a thin muscle bundle traversing the outflow tract of the left ventricle, attached to the base of the anterior papillary muscle and the septum beneath the aortic valve. At the base of the left ventricle there was a cylindrical bundle of muscle extending from the aortic cusps of the mitral valve along the anterior wall of the ventricle to the septum; there was thinning of the wall of the ventricle at the apex below this bundle. The ventricular septum was hypertrophied. The lungs were congested, with diffuse interstitial scarring, and there was passive congestion of the liver. Microscopical examination of the myocardium showed widespread focal scarring, with compensatory hypertrophy of the remaining muscle bundles.

The history and physical examination in this case presented features that were entirely consistent with rheumatic mitral stenosis. At operation and at autopsy it was apparent that a congenital anomaly of the myocardium of the left ventricle was producing effective stenosis of the ventricle that could not have been surgically corrected.

In retrospect the absence of history of rheumatic fever, the presence of an unexplained systolic "scratch" unlike that heard in mitral insufficiency and the electrocardiographic pattern of strain of the right and left ventricles might have caused the diagnosis to be suspected.

Case 3. J. B. (J.H.H. 664434), a 26-year-old truck driver, was admitted to the hospital on February 12, 1954, complaining of shortness of breath. The family and past histories were noncontributory. The patient had not been cyanotic at birth, and growth and development had been normal. He led an active, vigorous life in his first 17 years. There was no history of rheumatic fever. At the age of 17 he was rejected by the Army and Merchant Marine because of a heart murmur. During his 18th year he began to experience recurrent episodes of exertional dyspnea and paroxysmal cough, often with hemoptysis. At the age of 20 the dyspnea and hemoptysis became worse, requiring hospitalization in another institution, where he was placed in oxygen for 1 week. Orthopnea had become manifest at this stage, and at this time digitalization was first accomplished. He improved but continued to be somewhat dyspneic. At the age of 22 a prolonged episode of hemoptysis occurred, requiring 6 transfusions. Subsequently, the dyspnea, orthopnea and, in addition, intermittent nausea and vomiting persisted, although he continued to work as a truck driver. One month before admission he experienced abrupt dyspnea, orthopnea and cyanosis and for the first time noted ankle edema. Because these symptoms and signs failed to respond to diuretic therapy he was referred to the hospital.

Physical examination showed an acutely and chronically ill man in moderately severe respiratory distress who appeared younger than his stated age. The nail beds and mucous membranes were moderately cyanotic, and the neck veins distended. There was limitation of excursion of the left lower part of the thorax, and there were moist rales at both bases. The precordium was active. The point of maximal impulse was in the 5th interspace, 13 cm. from the left sternal border, and there was definite enlargement to the right as well. There was an apical systolic thrill. Auscultation revealed distinct accentuation of the mitral 1st sound, and at the apex there was a harsh, Grade 2 to 3, high-pitched systolic murmur. In addition there was a rumbling mid-diastolic apical murmur, with presystolic accentuation, heard best in the left lateral decubitus position. In the 3d and 4th interspaces, in the midprecordium, there was a 3d heart sound, interpreted by some observers as a protodiastolic gallop. In the same area a Grade 1, blowing systolic murmur was heard. The pulmonic 2d sound was loud and split. The liver edge was felt 7 cm. below the right costal margin. There was ++ bilateral pitting edema of the ankles.

The temperature was 98.8°F., the pulse 96, and the respirations 28. The blood pressure was 120/85.

Laboratory studies were not especially enlightening. The hematocrit was 49 per cent. The corrected sedimentation rate was 30 mm. per hour (Wintrobe method). The white-cell count was 5500, with a normal differential. Serologic tests for syphilis were negative, as were blood chemical studies except for a sodium of 131 milliequiv. per liter and a bilirubin of 1.5 mg. and a total serum protein of 7.2 gm. per 100 cc., with an albumin of 3.9 gm. and a globulin of 3.3 gm. Urinalysis showed a +++ test for albumin, with rare red cells and granular casts in the sediment.

An electrocardiogram showed strain of the right ventricle and digitalis effect. X-ray study of the chest revealed a markedly enlarged cardiac contour, in all diameters, with a prominent pulmonary conus. The cardiac margins were obscured by diffuse haziness, and the pulmonary vascular markings were increased, radiating from both hili. There was free fluid at both bases and in the fissures. Fluoroscopic examination was unsatisfactory because of the pulmonary congestion.

On admission the patient was placed on bed rest and a liquid diet containing 1.0 gm. of salt. Digitalis was administered in 0.2-gm. daily dosage, and mercaptomerin was given on several occasions. During his hospital stay of 4 days the liver seemed to decrease in size, but dyspnea, cyanosis and weakness increased. He was placed in oxygen, and a phlebotomy was performed, without relief. The pulse ranged from 90 to 110, and the temperature on 2 occasions spiked to 99.4 and 100°F. On the terminal hospital day atrial fibrillation developed. Intractable pulmonary edema persisted, and he died on February 16.

The final clinical diagnosis was rheumatic heart disease, with mitral stenosis and insufficiency.

Autopsy revealed extensive fibrous pleural adhesions, involving the parietal pericardium. There was 200 cc. of bloody pericardial fluid. The heart weighed 450 gm., and showed striking enlargement of the right atrium and ventricle. The left atrium and ventricle were normal, as were all the valves. There was dense tissue about the pulmonary veins, with marked constriction of all of these at the points of entrance into the left atrium (the orifice of the right superior vein was 2 mm., that of the right inferior vein 4 mm., that of the left superior vein 3 mm., and that of the left inferior vein 2 mm. in diameter). There was severe pulmonary arteriosclerosis; and the lungs were fibrous and granular. Microscopical sections of the dense mediastinal tissue demonstrated thick collagenous structure, with foci of degeneration and others of round-cell infiltration.

This patient gave a bizarre history of recurrent dyspnea, hemoptysis and sudden changes of clinical status, complicated by the appearance of severe cyanosis. The physical findings were compatible with mitral stenosis.

At autopsy it was found that the pulmonary veins were greatly constricted by a chronic collagenous process, which in effect created inflow obstruction into the left atrium. The exact cause of the murmurs is not clear, but they could have been on the basis of dilatation of the valve rings. If carefully performed x-ray study had been possible before death (especially if kymographic studies had been done and the left atrium could have been adequately visualized) this would probably not have been thought to be mitral stenosis.

Discussion

The diagnosis of mitral stenosis in most cases is relatively simple. It is well recognized that other disorders have certain clinical features in common with rheumatic disease of the mitral valve, but most of these entities can be recognized by careful clinical evaluation if alternative conditions are considered. However, in some cases, fortunately rather rare, the differentiation of mitral stenosis from other pathologic processes is not readily made and, in fact, can be exceedingly difficult.

In 1934 Waring and Black[1] discussed certain clinical features common to any process, including mitral stenosis, that obstructed the lesser circulation. The clinical findings proposed as comprising the "syndrome of obstruction in the lesser circulation" were cyanosis, dyspnea, polycythemia and hypertrophy of the right ventricle.

In 1935 Wyckoff and Bunim[2] emphasized the difficulty of differentiating certain varieties of lesser-circulation obstruction from mitral stenosis in some patients. They reported cases of primary pulmonary atherosclerosis, pulmonary schistosomiasis and chorionepithelioma that masqueraded clinically as rheumatic obstruction of the mitral valve.

In contrast Levine and Love[3] recently encouraged the recognition of mitral stenosis in the absence of murmurs. These authors described 19 cases of mitral stenosis in which, at one time or another, no diastolic murmur could be detected, and they estimated that this was true of 5 to 10 per cent of all cases of mitral stenosis. It was suggested that calcification of the mitral valve and "mitral" P waves in the electrocardiogram should be especially helpful in questionable cases.

Of the entities that clinically simulate mitral stenosis, intracardiac tumor, especially myxoma of the left atrium, can be most confusing. Physiologically, blood flow into the left ventricle is obstructed, leading to pulmonary hypertension and all the anatomic and physiologic alterations caused by true mitral stenosis. Auscultatory manifestations may be similar, as in Case 1 and as others have reported.[4-6]

The presence of tumor should be suspected when the murmur is inconstant or variable, when digitalis is ineffective early in the clinical course, in the absence of a history of rheumatic fever, by the appearance of rapidly progressive and severe decompensation, by the presence of severe dyspnea and precordial pain in episodes, by variations of murmur or clinical status with change of position or by any "atypical" features. Unfortunately, any one or all of these may be present in cases of clear-cut mitral stenosis, and the differentiation is not always possible. We have found angiocardiography helpful in demonstrating other intracardiac tumors.

In Case 1 the myxoma was successfully extirpated, and if embolism had not occurred the patient might have been "cured." This has suggested that in any patient with mitral stenosis in whom doubt exists, and perhaps in all subjected to commissurotomy, surgical means to extirpate a tumor should be available.

In Case 2 a myocardial anomaly functionally stenosed the outflow tract of the left ventricle; this must be a most unusual condition, and it was unprecedented in our experience. It seems doubtful if a correct diagnosis could have been made ante mortem—the nature of the alteration was not clear even at cardiotomy. This case was unlike congenital mitral atresia, in which there are usually a diminutive left ventricle, often overriding of the aorta, defects of the interatrial and interventricular septums, a patent ductus arteriosus and cyanosis.[7,8] It is doubtful if the usual case of congenital atresia would be mistaken clinically for rheumatic mitral stenosis but the question whether true valvular stenosis without other congenital defects can occur, or whether fetal endocarditis exists, is beyond the scope of this communication.

Case 3, in which constricting collagenous mediastinal thickening obstructed the pulmonary venous return to the left atrium, in many respects simulated mitral stenosis. A similar case, in which exploration was carried out by Dr. Alfred Blalock for possible

mitral stenosis,* as well as other cases of this unusual entity, is being reported in detail by Shulman and Harvey.[9] It is probable that, as in similar cases, adequate x-ray study would have diminished the clinical likelihood of mitral stenosis. The problem is similar to that of constrictive pericarditis, which, since mitral systolic and diastolic murmurs are not uncommon, has been often confused with rheumatic disease of the valve. Cardiac catheterization and kymographic study should allow adequate differentiation of these cases from true mitral stenosis, but cases in which constrictive pericarditis and mitral stenosis are thought to coexist will continue to be problems.

Many other pathologic conditions create auscultatory manifestations that in some ways simulate mitral stenosis. Congenital deformities of the heart are especially prone to the development of apical diastolic murmurs. The condition in which confusion is most likely to exist is defect of the atrial septum, which is sometimes present in association with a mitral diastolic murmur[10] and may be mistaken for Lutembacher's syndrome in the absence of organic mitral-valve deformity.[12] Patent ductus arteriosus, pure pulmonic stenosis, Eisenmenger's complex, coarctation of the aorta and defect of the ventricular septum have all been described as being accompanied, in some cases, by apical diastolic murmurs.[10,11]

Dilatation of the left ventricle can alter intracardiac dynamics in such a manner as to create relative mitral stenosis and thus to produce apical diastolic murmurs. This occurs in rheumatic carditis[13] and can lead to misinterpretation as structural mitral-valve disease. Anemia of any variety can cause relative ventricular dilatation and torrential flow, and mimic mitral stenosis on this basis; this is particularly true in sickle-cell anemia, which may readily lead to mistaken interpretation in view of the associated history of polyarthralgia that is commonly given in this condition.[14] Other causes of dilatation of the ventricle such as heart failure and a wide variety of organic heart lesions, including hypertension,[15] have been noted to produce apical diastolic murmurs. In pure mitral stenosis the left ventricle is not enlarged.

Any disorder of the lesser circulation may create physiologic alterations that are in some ways analogous to mitral stenosis. Apical diastolic murmurs may be audible in such cases,[1,2] but the clinical differentiation is usually not difficult. The problem of establishing the diagnosis of organic mitral stenosis in the presence of aortic insufficiency (Austin Flint[16]) is well known and need not be repeated here. It is common knowledge that the murmur of aortic insufficiency itself may be transmitted to the apex. Verrucous endocarditis in disseminated lupus erythematosus seldom leads to the clinical diagnosis of rheumatic mitral stenosis, although it is often interpreted as mitral insufficiency. Pectus excavatum and other thoracic-cage deformities may be associated with various heart murmurs, including some heard at the apex in diastole.

A final word regarding the mistaken diagnosis of mitral stenosis in totally normal hearts is warranted. Alimurung, Rappaport and Sprague have demonstrated phonocardiographically that some persons with structurally normal valves and without other features known to cause mitral diastolic murmurs may have alterations of the apical first sound simulating the presystolic murmur of mitral stenosis.[17] They suggest that in patients in whom only a presystolic murmur is heard, and especially when this is short and faint, phonocardiographic study may be most helpful in establishing the correct diagnosis.

It is apparent that the list of entities that can simulate rheumatic mitral stenosis is a long one. It is the responsibility of every physician involved in selection of candidates for commissurotomy to remain constantly aware of the difficulty of accurate diagnosis, and to be ever cognizant of the alternative possibilities even when "typical" signs of mitral stenosis are present. When atypical features are noted, it behooves the cardiologist to utilize all the potentially helpful accessory means at his command, including cardiac catheterization, angiocardiography, kymography, and perhaps ballistocardiography, to elucidate, so far as possible, the exact anatomic diagnosis before operation. When doubt still exists exploratory cardiotomy is warranted, after full explanation to the patient or his family, and in most cases is indicated, to give the patient every opportunity for surgical correction of the lesion if it is operable.

Summary and Conclusions

Three cases in which the clinical diagnosis of rheumatic mitral stenosis was made either preoperatively or ante mortem are reported. These patients were found actually to be suffering from the consequences of a myxoma of the left atrium, a congenital anomaly of the myocardium of the left ventricle and mediastinal collagenosis.

A brief review of the entities that may simulate mitral stenosis is presented, with suggestions to help differentiate some of these from true rheumatic disease of the mitral valve.

In view of the widespread application of commissurotomy in treatment of mitral stenosis care in the preoperative selection of patients is urged, as well as constant awareness of the multiple disorders that may imitate disease of the mitral valve.

Exploratory cardiotomy is advisable in cases in which obstruction seems to exist even though a definitive clinical diagnosis is impossible. Surgical facili-

*S. V. (J.H.H. 600747) was a 42-year-old housewife with a history of recurrent hemoptysis and dyspnea of ten years' duration. Examination showed cardiomegaly, with an accentuated pulmonic 2d and a prolonged mitral 1st sound, a short systolic apical murmur, but no murmur in diastole. X-ray study showed enlargement of the right atrium and ventricle and a mediastinal mass, which was thought possibly to be an enlarged pulmonary artery. An electrocardiogram was consistent with strain of the right ventricle. The pulmonary-artery pressure was 82/45, and the pulmonary "capillary" pressure 35 mm. of mercury (determined by Dr. Lewis Dexter). Examiners from Boston and Baltimore thought that mitral stenosis could not be excluded, and for that reason exploration was performed. At operation and autopsy extensive mediastinal and pulmonary collagenosis, with a normal mitral valve, was found.

ties for removal of tumor, commissurotomy, pericardiectomy, correction of a congenital deformity or other measures should be available.

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ELEVATED GLUCOSE THRESHOLD IN KIMMELSTIEL–WILSON SYNDROME*

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PATIENTS with intercapillary glomerulosclerosis often have an elevated renal threshold for sugar. This was noticed in 2 elderly diabetic patients with Kimmelstiel–Wilson syndrome who persistently showed markedly elevated blood sugar levels in the absence of glycosuria. The records of all cases of intercapillary glomerulosclerosis proved at autopsy at the Mary Hitchcock Memorial Hospital, Hanover, New Hampshire, and the Veterans Administration Hospital, White River Junction, Vermont, have therefore been investigated from this point of view, and the results indicate that there is a statistically significant elevation in the threshold in these patients as compared with other diabetic patients without the glomerular lesion. The implication of this finding is that such patients often have greatly elevated blood sugar levels without their physician's knowledge when control is maintained solely by means of tests for reducing substances in the urine. Whether or not this prolonged hyperglycemia contributes to the development of degenerative vascular complications of the diabetic state awaits statistical study of a large series of cases of Kimmelstiel–Wilson syndrome.

This series resembles the majority of reported cases[1-8] in that the patients were chiefly in the older age group and that their diabetes was of long standing. Eleven patients have shown intercapillary glomerulosclerosis at autopsy in the two hospitals since 1941. The average age at death was fifty-nine years, with a range of forty-six to seventy-two. This is close to the average age of 58 patients who died at the two hospitals in a comparable period with diabetes but no intercapillary glomerulosclerosis, the average age being sixty-five years. The known duration of diabetes in the group with intercapillary glomerulosclerosis averaged eighteen years—more than twice as long as the eight-year average of a comparable group of 17 elderly diabetic patients without intercapillary glomerulosclerosis autopsied at the Veterans Hospital.

Seven of the 11 patients died with massive edema due to either cardiac or renal failure. The other 4 died of complications of arteriosclerosis—1 of mesenteric thrombosis, 2 of cerebral thrombosis, and the third of coronary thrombosis. Six patients had hypertension, with blood pressures over 160 systolic and 100 diastolic. In 4 the diabetes was always controlled by diet alone without insulin; 3 needed at some time between 20 and 40 units of insulin daily, and the other 4 required up to 40 units of insulin or above. The 3 youngest patients, all men in their forties, were extremely difficult to control, their insulin requirements often varying wildly and unpredictably even on a constant diet in the hospital. They had extreme fluctuations in blood sugar levels from day to day, and excreted sugar readily as compared with the older patients, whose blood sugars were relatively stable and whose insulin requirements, as a rule, tended to decrease as the disease progressed. Glycosuria became less and less frequent in spite of reduced doses of insulin. Fasting blood sugars in many cases tended to be normal, but sugar was not excreted even when levels were high. The cases reported below illustrate these points.

Case Reports

The first case is one of decreasing blood sugar levels and disappearing glycosuria in a patient with a prolonged course.

CASE 1. L.H., a 55-year-old machine-tool worker, was found to have a ++++ test for sugar in the urine and a

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fasting blood sugar of 166 mg. per 100 cc. He was given a diet and advised to take 20 units of protamine zinc insulin daily. Six years later he presented himself at the hospital with the story that he had discontinued taking insulin after several months because his daily urine tests ran from negative to a trace. He complained of exertional dyspnea and failing vision, and had severe edema of the legs and retinitis proliferans. Glycosuria had disappeared, and a glucose tolerance test showed no sugar in the urine specimen at a blood level of 229 mg. per 100 cc. There was persistent albuminuria. The serum protein totaled 5.7 gm. per 100 cc., with 2.7 gm. of albumin and 3.0 gm. of globulin. Leg edema was relieved briefly by the insertion of Southey tubes, and for 2 years the patient spent most of his time at the hospital, where various combinations of diuretics were progressively less effective in relieving the congestive heart failure. A carcinoma of the right lung finally developed and he died at the age of 65. Autopsy revealed severe arteriosclerosis of the kidneys and intercapillary glomerulosclerosis. There was no sugar in the daily morning urinalysis for the last 6 months of life, and a fasting blood sugar toward the end was 102 mg. per 100 cc. He had not taken insulin at any time for 9 years before he died.

The next case illustrates that blood sugar levels may be very high in these patients in the absence of glycosuria.

CASE 2. I.S., a 55-year-old housewife, had weight loss and thirst and was put on 15 units of protamine zinc insulin to control glycosuria. She did well until 9 years later, when her husband died and she was overcome with grief, started drinking and neglected her diabetes. A year later she had albuminuria, a ++++ test for sugar in the urine and a fasting blood sugar of 400 mg. per 100 cc. In spite of her doctor's advice she ate indiscriminately and drank to excess. She later fell and fractured her hip, and on admission had an enlarged heart, ankle edema and rales at both bases. On this occasion the fasting blood sugar was 280 mg. per 100 cc., and the urine was negative for glucose. Six months later she was seen in the Outpatient Department with generalized anasarca, an enlarged liver, a ++++ test for sugar in the urine and 0.5 gm. of albumin per 100 cc. of urine. However, she refused hospitalization and when next seen was even more markedly edematous. Treatment with digitalis and mercurial diuretics was begun, and an effective diuresis, with a 20-pound weight loss was obtained over the next 2 weeks. The urine became sugar free, and the insulin dosage was leveled off at 20 units of NPH. Diarrhea persisted and she became extremely dehydrated and comatose. Blood chemical constituents on the day of death were as follows: blood sugar, 536 mg. per 100 cc., with no glycosuria (an intravenous drip of 5 per cent glucose was running at the time); carbon dioxide of 18 vol. per cent and sodium of 126 milliequiv. and potassium of 9.2 milliequiv. per liter. In spite of vigorous treatment she died 7 hours later. Control was difficult because of her neglect of advice and of failure to realize that the high glucose threshold prevented hyperglycemia from being reflected by glycosuria.

DISCUSSION

It will be noted that both these patients showed the phenomenon of absence of glycosuria in spite of elevation of the blood sugar. Table 1 demonstrates what I have termed the renal threshold for sugar in such cases. When glucose tolerance tests were not performed this had to be determined roughly by the highest blood sugar level obtained on a morning when the patient's urinalysis failed to reveal sugar. The normal renal threshold for glucose is usually given as between 140 and 190 mg. per 100 cc. of venous blood.[5] It will be seen that the average in the 10 patients in whom a threshold could be determined was 225 mg. per 100 cc. Since these figures simply show the highest blood values that were found on days when there was no sugar in the morning urine specimen, it is possible that the thresholds for sugar were appreciably higher. The table also discloses, in contrast, the similarly derived threshold for glycosuria in 13 elderly diabetic patients without intercapillary glomerulosclerosis, whose blood sugar level averaged 161 mg. per 100 cc., or within normal limits. Statistical evaluation of these data indicates that the ratio of the difference between these means to the standard error of their difference is 2.9, or that the likelihood that the samples varied to this degree by random chance alone is less than 1 in 100 times.

This phenomenon of a high renal threshold in intercapillary glomerulosclerosis has been observed by

TABLE 1. *Renal Threshold for Glucose.*

PATIENT	DIAGNOSIS OF INTERCAPILLARY GLOMERULOSCLEROSIS	PATIENT	DIAGNOSIS OF DIABETES WITHOUT INTERCAPILLARY GLOMERULOSCLEROSIS
	BLOOD SUGAR LEVEL *mg./100 cc.*		BLOOD SUGAR LEVEL *mg./100 cc.*
S.M.	374	D.D.	245
I.S.	286	J.H.	212
L.H.	229	T.G.	175
E.B.	218	C.B.	175
C.B.	209	L.W.	172
E.R.	208	N.B.	170
H.Ba.	206	R.H.	165
A.F.	202	A.P.	163
H.Be.	187	E.R.	153
C.S.	133	J.L.	134
		M.L.	126
		H.M.	113
		W.A.	101
Means*	225		162

*Difference between means, 63.2; standard error of difference, 21.6; level of confidence (P),[4] 0.01.

Rogers, Robbins and Jeghers,[3] who found that nearly half their 100 patients had minimal amounts of glycosuria and at the same time had blood sugar levels ranging up to 500 mg. per 100 cc. In 28 cases the blood sugar was 200 mg. per 100 cc. or over at the time glycosuria was minimal. The authors concluded, however, that it was impossible to state from their data whether or not elevated blood sugar with minimal glycosuria is of significance in aiding the clinical diagnosis of intercapillary glomerulosclerosis.

A physiologic explanation of why renal thresholds should be elevated in patients with diabetic intercapillary glomerulosclerosis is offered by the work of Corcoran, Taylor and Page,[6] who reported that these patients have a decreased glomerular filtration rate, renal plasma flow and maximum tubular capacity for glucose resorption. Rifkin et al.[2] likewise observed a reduced glomerular filtration rate, and Earle,[7] in a series of several patients, found a decrease in the filtration rate, the renal plasma flow, the maximum tubular resorption capacity for glucose and the filtration fraction. These findings indicate that the principal functional defect is in the glomerulus, as one would suspect. Renal plasma flow and glomerular filtration rate are so decreased that even with a de-

crease in ability to reabsorb glucose into the blood stream, the tubules can easily return the amount that is presented. Not until the blood sugar rises high enough for the concentration of glucose in the filtrate to exceed the load the tubules can reabsorb will glycosuria occur. This can only be accomplished by elevation in the concentration of glucose in the blood stream and hence in the glomerular filtrate.

A possible explanation why average blood sugar levels should, over a long period, gradually fall in older patients with intercapillary glomerulosclerosis is presented in the work of Elgee and his colleagues,[8] working with radioactive insulin. In normal rats the greatest concentration of radioactive insulin was in the renal cortex—specifically, in the convoluted tubules according to radioautographs. Apparently, insulin is metabolized in the kidney. Insulin breakdown was greatly impaired by nephrectomy. Forsham, in discussing the paper, suggested that these findings may explain increased insulin sensitivity in advanced Kimmelstiel–Wilson syndrome. Certainly, there is tubular as well as glomerular damage, as shown both by histologic examination[2] and by kidney-function studies.

Disappearance of sugar for long periods from the urine of an elderly diabetic patient with intercapillary glomerulosclerosis may be due to an elevated renal threshold or to increased insulin sensitivity, or to both. A glucose tolerance test should be performed, and if the threshold is high, the subsequent absence of glycosuria will not be wrongly interpreted as indicating absence of hyperglycemia.

Summary and Conclusions

A group of 10 patients with Kimmelstiel–Wilson syndrome is described. These patients averaged sixty years of age at death, and had had their diabetes for approximately twice as long as a comparable series of diabetic patients without intercapillary glomerulosclerosis. The clinical course of 2 patients is described, and it is shown that a characteristic feature of the group appears to be an increase in the renal threshold for glucose. This results from a decrease in renal plasma flow and glomerular filtration rate. It may result in unrecognized hyperglycemia if patients are followed only by tests for glycosuria.

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MEDICAL PROGRESS

PROCTOLOGY

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IN attempting to select articles from recent surgical publications for inclusion in a progress report on proctology one is immediately confronted with the necessity of deciding what areas this term properly includes and what subjects are of greatest importance and interest. There are specialists in proctology who confine their work to diagnostic procedures and minor surgery of the anal canal and rectum only. Others, with training and the desire to do major surgery of the colon and rectum, include this wider field in the scope of their activity. Then there is the large group of general surgeons who do a great deal of the rectal and colonic surgery that is done, often having a special interest in it, but not limiting their surgery to this field.

A recent Medical Progress report on abdominal surgery by Welch[1] included about 30 articles on large-bowel conditions, most of which I have not repeated in this review. The reader is also referred to a prior review by Turell, Krakauer and Maynard[2] on this general subject.

Except for a few case reports of unusual conditions this review makes no attempt to cover the many articles on large-bowel carcinoma and its surgical treatment that comprise about one third of the publications listed under the general heading of the rectum and colon in the *Current List of Literature of the Armed Forces Medical Library.* I have also eliminated by title, or on casual inspection, many contributions on other subjects that did not give promise of worthwhile content, and I have made no attempt to summarize foreign-language articles. A selection has been made from papers presented before the American Proctologic Society, together with others of special interest or unusual nature.

The most common pathologic conditions encountered in this field are hemorrhoids, fissures, fistulas, and pruritus. So much has been written about these conditions in the past and the surgical treatment of the first three is so well standardized, and usually so

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simple, that not many articles on these common anal conditions have recently appeared.

Pruritus Ani

Pruritus ani continues to be a frequent and distressing ailment, the cause and cure of which are not known and the remedies for which are legion. With what is essentially a dermatologic problem these patients come under the care of specialists in both fields, going from one to the other as they see little progress toward permanent cure. Every general practitioner quite properly undertakes to treat these patients also.

Many unnecessary anal operations are performed on people whose only complaint is itching because the surgeon is willing to attribute this symptom to one or more of the various abnormalities of the anal canal that sometimes, but by no means always, coexist. Cleanliness and various washes and ointments give relief. Occasionally, one seems to cure a patient, but this is a rare, and seldom authenticated, occurrence. Conscientious research on this subject, if successful, would bring relief to victims so numerous as to eclipse those suffering from heart disease and other more widely publicized and admittedly more serious conditions. There is no real progress to report.

Hemorrhoids

Well conceived and properly executed surgery continues to be the only method by which internal and external hemorrhoids can be completely and permanently removed (Dr. Brady's syndicated column in the newspapers "to the contrary notwithstanding"). The injection of sclerosing solutions, however, which is an excellent method of eliminating the bleeding of internal hemorrhoids and the protrusion of all except the largest, for limited periods, can always be utilized for those who wish to avoid surgery, provided they understand its limitations as well as its certain advantages.

The possibility of pulmonary emboli as a result of thrombosis in the hemorrhoidal veins, either spontaneous or after the injection of internal hemorrhoids with sclerosing solutions, has been discussed from time to time, but I have never seen it myself and never talked with anyone who had seen it occur, nor have I seen a report in the literature on this subject, until now.

Stone and Lovelock[3] report a case of multiple pulmonary infarction occurring in a thirty-three-year-old Negro who had been ambulatory up to the time of onset. Several episodes of sudden chest pain with slight hemoptysis had occurred before he was hospitalized. On admission there were no leg signs whatsoever — a negative Homans sign and no calf tenderness. There was a ring of tender external anal skin tags, and within the rectum a nodular, tender area posteriorly above the sphincters.

Four days later the patient had another episode of severe pain in the left side of the chest, with bloody sputum, normal temperature, a pulse of 100 and apprehension. X-ray study showed slight pleural effusion on the left and a definite circumscribed shadow at the base of the left lung. Examination of the leg veins was still negative but the area in the rectum was much more tender and the temperature rose to 102°F. for several days. All sputum examinations were negative.

Proctoscopic inspection revealed a proctitis, with ulcerations on the lower 6 cm. of the rectal mucosa and other ulcerations at 12 cm. A Frei test was positive, and with a low proctitis the diagnosis of lymphopathia venereum seemed justified, although the patient had no inguinal adenitis and could recollect no recent penile lesion. After treatment with sulfadiazine for a month, and when proctoscoped after seventy days, the ulcerations had healed.

In favor of the possibility that this represented a case of pulmonary embolism originating in the hemorrhoidal veins, in the presence of adjacent infection, the authors cite the following considerations:

1. The inferior and middle hemorrhoidal veins drain into the internal pudendal and hypogastric veins and the internal iliac, though also anastomosing freely with the portal system above. Systemic embolism should therefore be possible.
2. In a report by J. King, two of ten cases of pulmonary embolism occurred in soldiers with acute thrombosed hemorrhoids, and phlebography of their leg veins was negative. T. J. Moran reported three cases in which, based on autopsy findings, he felt the emboli had come from the hemorrhoidal veins, although in these three cases accurate details as to autopsy findings were not given.
3. Failure to implicate the rectum as a source of emboli may well be due to the frequent omission of an examination of the veins of this area by pathologists. In certain reported series of cases of pulmonary embolism no source has been found in about a third of the cases.
4. Phlebography, however, is not always reliable when positive or when negative, as brought out by A. W. Allen.
5. Although the case herein reported did not have phlebograms made, the possibility that the emboli did in fact come from thromboses of the hemorrhoidal veins in the presence of lymphopathia venereum deserves consideration.

Gerwig and his associates[4] have sought to eliminate pain after hemorrhoidectomy by a method of continuous caudal anesthesia, carried out for forty-eight hours after operation. Using an 18-gauge, 6-cm., thin-walled, caudal needle, they insert through its lumen, for 6 cm. into the anal canal, the end of a 60-cm. piece of vinyl tubing. Then the caudal needle is withdrawn, and a blunt, 23-gauge needle is inserted into the distal end of the plastic tube and a hub adapter added.

To produce anesthesia for the operation an initial injection of 30 cc. of 0.15 per cent tetracaine hydrochloride is given, and this is repeated in 20-cc. doses, varying from every three up to every six or seven hours, for forty-eight hours after operation, thus producing a continuous freedom from pain for that pe-

riod. This procedure was carried out in 59 cases with no infection.

The authors are to be commended for their efforts to reduce pain after surgery of the anal area, which has always had a reputation for being attended by considerable pain. There are, however, certain facts regarding this subject of whose truth considerable experience has convinced me. In the first place there is great individual variation in complaints of pain. Also, the immediate postoperative pain can be well controlled by morphine given promptly and in adequate dosage, and the severity has greatly subsided after a period of five to eight hours. The majority of patients complain very little (sometimes not at all) on the first day after operation. On the second day any complaint of pain or discomfort is more likely to relate to the abdomen — a feeling of unrest, the rolling of gas and so forth — than to the anal area. The first bowel evacuation, commonly the high point in postoperative discomfort, usually occurs on the third day, which would be after the forty-eight-hour period during which the authors use the tetracaine injection. In this event one wonders if the complete absence of pain for forty-eight hours would make the discomfort encountered thereafter seem worse than if the patient had already experienced what might be called a "normal" amount of postoperative pain. Is the prolonged use of tetracaine (or a similar drug) in nerve-block anesthesia safe enough from possible complications to justify its administration routinely? Could any permanent nerve damage result from it? These are all factors to be considered although the authors have apparently encountered no ill effects in their experimental series of cases, and this procedure might be expected to give more adequate relief of pain than perianal injections of oil-soluble anesthetics.

Granet[5] describes a procedure for removal of internal and external hemorrhoids that he considers to be the answer to the problem of a complete removal of extensive, annular hemorrhoidal involvement without such resulting complications as stricture, residual piles, mucosal prolapse, pseudopolyps and tags. Using a wide quadrilateral posterior incision, he dissects forward, beneath the skin and the superficial sphincter fibers, on up into the anal canal until he reaches normal mucosa beyond the internal hemorrhoids of the entire posterior half of the circumference of the low rectum. He then cuts transversely through mucosa, which is held with Allis clamps. After any arterial bleeders have been tied beneath this mucosal pedicle its free edge is sutured to the border of the deep sphincter at about where the dentate line would normally lie. Small anterolateral varices are then excised subcutaneously, through small incisions in these quadrants, and the large right anterior internoexternal hemorrhoid is excised and ligated individually and its stump sutured to the subcutaneous tissues at the anal verge. The bowels are moved on the third postoperative day by means of hydrophilic laxatives started soon after operation and continued thereafter. Cleanliness is achieved by a warm tap-water spray rather than sitz baths, the patient sitting over the edge of a tub. Granet follows his patients weekly for six weeks, and at intervals for six months, and considers his results to be consistently satisfactory.

This article is an attempt to solve the problems of how to achieve an adequate, yet not too radical, removal in the cases in which hemorrhoids seem to occupy the entire circumference of the anal canal and anal outlet. This situation confronts every surgeon from time to time and demands considerable restraint to avoid the serious error of removing too much skin in an effort to obtain a good immediate cosmetic result. I question somewhat the wisdom of a procedure that, at the outset, reduces the perianal skin and anal-canal lining by half, in spite of the apparent care with which Granet deals with the remaining anterior half. It has been my experience that, even in these cases with complicated and extensive involvement, a judicious selection of areas of removal using elliptical incisions and varying the amount of skin removed according to its degree of redundancy will accomplish a satisfactory functional and cosmetic result even if the appearance of the anal outlet at the end of the operation is not that which a plastic surgeon might hope to attain. It is surprising how nearly normal in appearance an end result will be after complete healing has ended the persistence of edema in the adjacent skin.

Prolapse

Gabriel[6] has summarized the revived interest in Thiersch's operation for anal incontinence in a report of 25 personal cases with no mortality and no serious complications. The procedure consists of the introduction of a single No. 19 or 20 silver wire so as to encircle the patulous anal canal, utilizing a Doyen needle and working through 0.6-cm. incisions anterior and posterior to the anus. The wire is passed so that the two ends protrude from the posterior incision. It is then tightened on a forefinger inserted full length through the anal canal, thus producing a wire circle about 2.5 cm. in diameter. After the ends have been twisted to secure a firm binding and the excess wire cut away the twist is pushed into the posterior incision and buried in the deeper tissues by being bent posteriorly.

The two skin incisions are then closed, the entire operation having been carried out with careful regard for asepsis.

Gabriel emphasizes the fact that the Thiersch operation is not applicable to large, complete prolapses of the rectum. He has used it in 25 women, 7 of whom were spinsters. Of the remaining 18, 5 had never borne children whereas 13 had borne one or more. Seventeen were over sixty, and 9 over seventy years of age. These patients fell into three general groups, as follows: primary anal incontinence (relaxed

sphincter with mucosal prolapse or a short, complete prolapse), 16 cases; secondary anal incontinence (relaxed sphincter with mucosal prolapse after previous major operations for complete procidentia), 8 cases; and anal trauma (after a fistula operation with complete division of the sphincter), 1 case.

Postoperative care included efforts to keep the area clean and dry, avoidance of impaction by use of mineral oil orally, and glycerin suppositories.

Complications consisted of an occasional breaking or untwisting of the wire and of ulceration through the skin. In each case the wire should be removed; it can be replaced at a later date.

Results were considered satisfactory in 21 of the 25 patients, 6 of whom required one or more additional operations. There is a greater likelihood of sepsis in repeated insertions of the wire, which should be delayed until some time after complete healing of the incisions has occurred.

Berkowitz,[7] in Boston, has reported on the Thiersch operation in 3 women, seventy-five, seventy and sixty-eight years of age, under local novocain anesthesia in lithotomy position. These women all had a rectal procidentia 8 to 15 cm. in length, and the result was satisfactory for short follow-up periods of ten, nine, and four months. Berkowitz used two 13-gauge, straight, hollow needles inserted through an incision posterior to the anus and emerging through an anterior incision, one needle going up each side of the anal canal. One end of a No. 22 stainless-steel wire was then threaded into the point of each needle, emerging through the base of the needles, which were then withdrawn and the wire ends twisted and cut as described by Gabriel.[6] Immediate ambulation was allowed, and there were no complications.

It will be of interest to learn how long a period of relief is obtained in these 3 patients, all of whom had a sizeable, complete prolapse; a fairly early recurrence by breakage of the wire seems inevitable because of the size and nature of the original prolapses, as indicated in the preceding report of Gabriel.[6]

The question of how much the sigmoid may prolapse into, or intussuscept, the upper rectum and what, if any, symptoms result from this situation of "concealed prolapse" has been difficult to evaluate. That it can occur in some degree is suggested by observation during proctoscopic examination with the patient in lithotomy or semiprone position (neither of which is recommended for proctoscopy as a rule). Also, by digital examination, one can sometimes feel at the finger tip a rounded protrusion of contracted low sigmoid into the upper end of the rectal ampulla. I have no doubt that this sort of presenting sigmoidal prolapse has been thought to be a tumor in an occasional patient in whom subsequent investigation has disclosed none.

Granet[8] considers this sort of prolapse to be a normal physiologic occurrence with the defecatory reflex, varying with the length and mobility of the mesosigmoid and reducing itself spontaneously. The small caliber of the sigmoid, the large caliber of the rectum and the fixation of the rectosigmoid area by the pelvirectal and sacral fascia favor this occurrence.

Symptoms attributed to this type of prolapse are a sense of incomplete and unsatisfactory evacuation after defecation that is accompanied by straining. This may produce a dragging sensation in the pelvis and low back and a dull ache in the perineum and thighs. Glairy mucus and occasionally blood may be passed.

Granet describes a special technic with which the presenting sigmoid can be shown by a small barium enema and air insufflation.

The regular use of psyllium hydrophilic preparations by mouth and an explanation to the patient of the mechanics of the prolapse, with assurance and avoidance of straining at stool, are usually adequate treatment.

Dunphy et al.[9] reported results in 17 operations for complete procidentia of the rectum with only 1 recurrence. Circular amputation was performed in 3 cases, in 2 of which an abdominal procedure, as described below, was later done. Twelve patients were treated by a combined operation as described by Dunphy in 1948. Through an incision suture of the levators was carried out after the anterior rectal wall and deep cul-de-sac had been freed and pushed upward. Then the abdomen was opened, and the pelvic colon mobilized, including a removal of the deep pelvic peritoneum. A Mikulicz resection of the redundant sigmoid loop was then carried out, and the pelvic floor closed at a higher level after suture of the transversalis fascia beneath. When the sigmoid proved to be short it was not resected but sutured to the lateral pelvic wall for support. This was done in 2 cases.

The pros and cons of these different procedures are thoroughly discussed. Dunphy believes that his combined operation offers more prospect of a long-term cure than any other method he has used.

Cysts

Bonser, Raper and Shucksmith[10] reported 4 cases of epidermoid cysts of the rectum and anus seen over a four-year period and referred to 21 cases previously reported by others. Of the 4 cases a fistula was present and communicated with the cyst in 3. Multiple cysts behind and alongside the rectum were present in 1 case. Of the 21 cases previously reported only 3 had established a fistula. It is the opinion of the authors of this article that these cysts can be regarded as teratomatous because of the findings of more than one type of epithelium in all 4 cases.

Congenital Malformations

Congenital anomalies of the anus and rectum are rare in the experience of most proctologists and general surgeons, the majority of these patients presumably being seen and handled by surgeons doing pediatric surgery. Reference to the book by Ladd

and Gross discloses most of the clinical experience with these conditions in New England.

Bacon and Sherman,[11] of Philadelphia, reported an experience with 111 cases of congenital anorectal deformities in 63 male and 48 female infants, of whom 32, or 29 per cent, had associated congenital anomalies of one sort or another. Of the 111 patients 47 had anal anomalies. There were 3 deaths on admission, and all but 1 of the remaining patients were operated on, with 3 postoperative deaths.

The procedures included simple incision and dilatation in 15, and anoplasties in 7 cases of stenosis of the anal canal in which the anal orifice was present. In addition there were 22 patients with no anal orifice. In 8 of these, having a membranous occlusion only, incision and dilatation only were required, whereas in 15, who had a mural or fibrous occlusion, small plastic procedures were required, including the Rizzoli technic to open and transplant the closed anal outlet from the perineum to its normal position between the ischial tuberosities.

This series embraced 64 rectal, as contrasted to 47 anal, anomalies. In 40 of these there were fistulas, 29 of which were rectovaginal. The Rizzoli method was quite satisfactory in correcting the latter but in the remainder an abdominoperineal (pull-through) procedure was carried out, usually at three to five years of age, after a transverse colostomy for relief of the obstruction at birth.

The over-all mortality for 108 patients treated surgically was 6.4 per cent.

Perforations

Klein and Scarborough,[12] of San Francisco, reported 50 cases of traumatic perforations of the rectum and distal colon, from a variety of causes and with an over-all mortality of 10 per cent. Of 11 perforations during proctoscopic examinations all occurred in the hands of inexperienced doctors. Eight were recognized during the proctoscopic procedure, and a laparotomy was performed within two to four hours, with 7 uneventful recoveries and 1 death on the eleventh day from peritonitis and cardiorenal disease in an elderly woman. The remaining 3 perforations were unrecognized for over eighteen hours, or until signs of peritonitis had appeared, and 2 of the 3 patients died. In 10 of the 11 cases the perforation was intraperitoneal.

An analysis of the probable technical errors that caused these perforations included the following: blind introduction of the proctoscope beyond the anal canal, reinsertion of the obturator to overcome "spasm," attempted forceful dilatation of a rectal stricture, careless use of long, cotton-tipped applicators, excessive fulguration of a rectal polyp and biopsy through the bowel wall at the border of a carcinoma.

Perforations during Enemas

In this group, there were 17 cases, in 5 of which the perforation occurred during rectal enemas, whereas in 12 it occurred during colostomy irrigations. There were no deaths among the 5 patients with perforations during rectal enemas. The bowel wall was normal in 4. The perforation was intraperitoneal in 3 and extraperitoneal in 2.

Perforation during colostomy enemas in 12 patients produced pain around the colostomy and evidence of peritoneal irritation within several hours and a localized inflammatory mass around the colostomy in 7 cases treated conservatively. Laparotomy in the other 5 consisted of simple closure of the perforation in 2 cases, a transverse colostomy in 2 and simple laparotomy with drainage in 1 case in which the hole could not be located because of inflammatory adhesions.

Perforations from Other Causes

This series also included 5 patients whose perforations occurred from impalement on, or the forcible introduction of, long, sharp objects per rectum, 11 others perforated by gunshot or knife wounds or other forms of external trauma, and 6 patients in whom the injury occurred during surgical operations on adjacent organs.

Principles of Treatment

Immediate laparotomy is essential in perforations by the proctoscope. Suture of an intraperitoneal perforation without a proximal colostomy is the procedure of choice unless the trauma appears to have caused devitalization of bowel wall, when a colostomy in addition to closure is desirable.

When the perforation is extraperitoneal a colostomy should always be performed. The extrarectal infected area will presumably drain back into the bowel through the perforation itself and will eventually heal spontaneously after diversion of the fecal stream by colostomy, aided by the use of sulfonamides and antibiotics.

Gas Explosions

Becker[13] cites several reports of serious intrarectal explosions during electrocoagulation of polyps and offers suggestions to avoid this risk. Multiple perforations of the colon from this cause have been reported by Montier, of Paris, in 2 cases, necessitating laparotomy. Liebermann has reported several explosions of lesser degree.

Intestinal gases expelled by man, as analyzed by Fries, were found to consist of carbon dioxide, 10.3 per cent by volume, oxygen, 0.7 per cent, methane, 29.6 per cent, and nitrogen, 59.4 per cent. Other factors concerned in explosions are the gases of outside air introduced through the proctoscope and gases resulting from the electrodesiccation of tissues.

Becker has devised short and long scopes through which carbon dioxide can be introduced from a tank equipped with a reducing valve, providing a steady flow over the intrarectal field of operation during electrodesiccation, which is thereby carried out in an atmosphere that does not support combustion.

I have personally never seen an intrarectal explosion occur, nor does Becker say that he has, but he is to be commended for giving thought and effort to the elimination of a hazard that has been reliably reported to exist and may at any time occur during high-frequency electrodesiccation within the bowel if inflammable gases are present in adequate concentration and with sufficient oxygen to favor ignition.

Pilonidal Disease

Swinton and Markee,[14] of the Lahey Clinic, reported the results of a questionnaire sent to all members of the American Proctologic Society concerning the treatment of pilonidal sinus. Replies from 248 surgeons (51.5 per cent of the members) covered a total experience of 25,320 cases. Of the 248 surgeons replying 70 per cent favored open operations with varying technics and 30 per cent used a closed operation in selected cases. In a report by Kleckner in 1936, based on a similar questionnaire and 4000 cases, open operation was favored by 87 per cent of those replying, and in a report by Rosser in 1947 the figure was 77 per cent, as compared with the present figure of 70 per cent. There is thus a definitely increasing trend toward fewer open and more closed operations. Also, this latest study revealed a trend away from massive excisions toward limited excisions, with partial closure or marsupialization of the cyst.

A study by Dwight,[15] of Boston, compiled from an experience with 245 patients seen during two years at the Cushing Veterans Administration Hospital in Framingham, condemns the use of the gluteal-flap method of primary closure. This operation was utilized in 135 cases, of which 13 were immediate failures because of sepsis. In 16 the incisions reopened within a year. There were less serious complications in 19 other cases. Although the results in the remainder were good, Dwight believes that the additional operations needed and the greatly prolonged convalescence in the patients who did not do well were too great a price to pay for the good results. In the successful cases, the healing time was a week or two, whereas in the 29 unsuccessful gluteal-flap cases, 80 per cent of which required one or more secondary operations, the healing time averaged fifteen weeks, the longest being six months. In a series reported in 1938 by Rogers and Dwight, and also in 53 cases in the present group treated by open operation, the average healing time was nine weeks. Dwight also cites a series by Knowles and Baker, who mobilized the gluteal flaps in 187 cases by cutting the gluteal fascia close to the sacrum. The procedure, whose rate of failure was 37 per cent, has also been abandoned by the authors.

As a result of his experience in this series and in previous ones Dwight has now adopted minimal excision and closure without plastic flaps in selected small midline lesions and open operation in all others.

In my own experience, which consists almost entirely of a limited but complete excision of cysts and sinuses, with suture of the wound edges to the base of the wound on each side, the patients leave the hospital in two or three days, with removal of the sutures in the office later and complete healing in two to four weeks, with occasional exceptions.

Benign Adenomas

The relation of adenomas of the rectum and colon to cancer of these areas is a continuous subject of discussion although no one seriously disputes the fact that benign polyps often become malignant.

Morton[16] has presented some figures based on 1237 consecutive proctoscopic examinations done by himself. All these patients were seen because of some complaint relating to the large bowel so that the incidence of adenomas and cancer was higher than it would have been in the same number of persons picked at random. From 1 to 9 adenomas were found in 129 (10.4 per cent) of these patients, and carcinoma of the rectum was observed in 55 (4.4 per cent). Carcinoma combined with one or more benign polyps was observed in 8 of the 55 (14.5 per cent), a figure quite close to the 18 per cent reported in a series of Jackman and Mayo.

Morton makes a plea for the institution of better facilities in hospitals for proctoscopic examinations and the training of all interns in this procedure. He urges the complete removal of polyps when feasible, preliminary biopsy being utilized only in the larger ones.

Welch, McKittrick and Behringer[17] made a clinical and pathological study at Massachusetts General Hospital of patients with single, or several adjacent, polyps of the colon and rectum with a view to correlating the surgeon's clinical judgment of the nature of the polyp with subsequent pathological findings. A series of 322 cases was accumulated from hospital records between 1937 and 1952, including follow-up studies in 165 of these from 1937 to 1947.

The 322 cases included 26 *papillary adenomas*—all in the rectum or low sigmoid. These tumors have a high incidence of malignant degeneration, and the authors believe that they should be attacked by a radical type of operation. Of the 26 patients 7 showed cancer microscopically, and 3 died of cancer. There were 21 radical and 5 local excisions, 2 of the 3 cancer deaths occurring in the latter group.

The remainder (296) of the 322 patients had *adenomatous polyps*—a less dangerous tumor but one that is also capable of malignant change.

Of the 322 cases, cancer was found in 60 (18.6 per cent), varying from "carcinoma in situ" to the higher grades of malignancy. Of the 60 patients 34 had local excisions of the polyp, and 9 of these recurred or developed metastases later — all 9 patients having had either a papillary adenoma or an adenomatous polyp with a high-grade cancer.

The authors advocate segmental resection of colonic polyps, rather than colotomy with polypectomy, in all

cases, as the surest way to avoid the necessity of a secondary resection in some cases and the surest way to eliminate local recurrences and any regional metastasis that may have occurred if the polyp should be found to be malignant.

Harris[18] has reported 70 cases of polyps of the colon and rectum observed in 56 male and 44 female children under twelve years of age during the past nine years in his own private practice. Removal was carried out in 60 of these, of whom only 12 required laparotomy. The locations of the polyps were as follows: rectum, in 58 patients; sigmoid, 16; and above the sigmoid, 6. Harris found only a single polyp in 52 patients, 2 in 7, 3 in 4 and 4 or more in 3 patients. Blood-tinged mucus was the commonest symptom, and diagnosis was easily made by the use of an adult-size proctoscope and a double-contrast barium enema. Spontaneous amputation of the polyp occurred in 8 definitely proved cases, the patients bringing the polyp for inspection in 4.

Removal of polyps accessible through a proctoscope was accomplished by electrocoagulation, using a snare below the peritoneal reflection and fulguration above the reflection. By the latter procedure a pathological examination was not always possible, but the incidence of malignant change in polyps in young children is so low that preservation of the polyp for microscopical section was not considered essential.

A point of special interest was the fact that whereas Harris found 70 children under the age of twelve with polyps he could locate none between the ages of twelve and nineteen. He raises the question whether, by twelve years of age, most polyps become self-amputated.

Briggs and Roberts[19] report 3 cases of gastrointestinal hemorrhage due to benign polyps of the duodenum. The bleeding in 2 cases was massive. Two of the tumors were adenomas of Brunner's glands, and the third originated from intestinal glands. Two were pedunculated, and 1 was sessile. These polyps are of interest because of the difficulty of x-ray demonstration and the absence of any symptoms except bleeding. Their removal was simple.

This brief report concerning polyps above the colon as a source of bleeding is inserted to call attention to the fact that the use of a sigmoidoscope and barium enema with negative findings, and even negative upper gastrointestinal x-ray study in addition, does not necessarily exclude the presence of polyps, which are more difficult to demonstrate in the small bowel than in the stomach or colon, and especially so in the duodenum.

(To be concluded)

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MEDICAL INTELLIGENCE

MASSACHUSETTS MEDICAL SOCIETY TOWNSHIP

THE following act of generosity is found in the *Resolves of the General Court of the Commonwealth of Massachusetts, Respecting the Sale of Eastern Lands; with the Reports of the Committees Appointed to Sell Said Lands; From February 2, 1804 to June 14, 1810.* Printed by Isaac Munroe, printers to the state, in Boston in 1811.

RESOLVE

Granting the Massachusetts Medical Society a township of land.

February 10, 1810.

WHEREAS the Massachusetts Medical Society have incurred expences, and have devoted a considerable portion of their time, to promotion of the laudable objects of their institution, and have petitioned this Legislature for some pecuniary aid, to enable them to erect a suitable building in the town of Boston, for the use of said society—Wherefore,

Be it resolved, That there be, and there hereby is appropriated for the use and benefit of the Massachusetts Medical Society, one township of land, to contain six miles square, and to be surveyed, located and assigned from any of the unappropriated lands belonging to this commonwealth in the district of Maine, (excepting the townships lately purchased of the Indians, and lands contracted for by Jackson and Flynt,) under the direction of the agents for the sale of eastern lands, at the expense of the said society: *Provided,* said location be made within three years, a plan whereof to be lodged in the land office; and the agents aforesaid, are hereby authorised and directed to give good and sufficient deed or deeds of the same to the Trustees of said society, or their assigns, subject to the usual reservations and conditions of settlement.

Not only was the General Court friendly to the young medical society in those days; it was prepared to show its friendliness at the expense of the future State of Maine. There is no record that the good intention was acted on within the prescribed three years.

CASE RECORDS
OF THE
MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

BENJAMIN CASTLEMAN, M.D., *Editor*
VIRGINIA W. TOWNE, *Assistant Editor*

CASE 40331

PRESENTATION OF CASE

A sixty-five-year-old businessman was admitted to the hospital because of fever and malaise.

The illness began two weeks previously, when flushing and nausea gradually developed. The patient felt very warm but had no pain. He remained on modified bed rest because of malaise. On the third day a "swollen, itchy, lobster-colored" skin rash developed that seemed to be improved by Benadryl. During this period he noted easy fatigability. On the seventh day of his illness he felt well enough to visit his summer home. On arrival he went to bed and was awakened at midnight by severe pain on both sides of the lower back, without radiation, and a shaking chill. The temperature was 103°F. and penicillin, chlortetracycline and a sulfonamide were given. The temperature continued to spike to 103°F. almost daily, and his skin began to flake. He had no urinary symptoms except hesitancy; the urine was described as clear and dark yellow. During much of this time his fluid intake was low, and he vomited frequently. He complained of low-back pain, malaise and nausea. The back pain was also "well to the sides" but did not radiate and had no relation to urination. During the day before admission he again had a shaking chill, with a temperature spiking to 103°F., and complained of pain in the right shoulder and left hip. On the day of admission he complained of bilateral costovertebral-angle pain, malaise and nausea. Because of the continued symptoms he was admitted to the hospital.

Sixteen years before admission he had been hospitalized for duodenal ulcer; since that time he had been free of ulcer symptoms. He had also been hospitalized for occlusion of the right brachial artery, acute enteritis, ligation and stripping of the right saphenous system, fourteen, three and two years before admission, respectively. The etiology of the brachial-artery occlusion was unknown. It was treated with dorsal sympathectomy on the right and section of the right anterior scalenus muscle. All pulsations in the legs were

present fifteen years before admission; during the hospitalizations after that time, however, the femoral and popliteal pulses were recorded as present, and the dorsalis-pedis and posterior-tibial pulses as absent. He gave a history of chest pain on exertion for five or six years before admission that was not progressive.

Physical examination revealed a well developed, well nourished man lying comfortably in bed. The skin was flushed and over the entire body was involved in a scaling rash. The chest was clear to percussion and auscultation; the heart was normal. No masses were found in the abdomen, but there was mild bilateral costovertebral-angle tenderness, with some tenderness over the left-kidney region on palpation anteriorly. The right brachial and radial pulses and both popliteal and dorsalis-pedis pulses were absent. Rectal and prostatic examinations were negative. There was no peripheral edema, and aside from varicose veins the legs were normal.

The temperature was 101°F., the pulse 95, and the respirations 23. The blood pressure was 100 systolic, 70 diastolic.

The urine on admission was normal except for a + test for albumin and 8 red cells and 3 white cells per high-power field in the sediment. Subsequent urinalyses were similar; the white cells and red cells in the sediment were never over 8 per high-power field. Examination of the blood showed a hemoglobin of 12.6 gm. per 100 cc. and white-cell count of 24,000, with 93 per cent neutrophils. On repeated examinations of the blood white-cell counts varied between 31,000 and 11,300, with about the same differential count. The bilirubin was less than 0.2 mg. per 100 cc. Agglutination reactions for brucella, typhoid H, typhoid O and paratyphoid A and B were negative. The Widal test was positive in a 1:160 dilution. Blood cultures were negative at forty-eight hours. Stool culture was negative for pathogens. Multiple urine cultures and one culture of prostatic fluid showed rare *Proteus vulgaris,* which was sensitive to chloramphenicol.

A roentgenogram of the chest revealed a prominent heart shadow in the region of the left ventricle. The aorta was moderately tortuous. A Graham test suggested impairment of function. Examination of the right femur and pelvis was negative; the dorsal lumbar spine showed only minimal degenerative changes. The abdominal aorta was calcified and tortuous (Fig. 1). The psoas-muscle shadows were not well seen, but they appeared grossly symmetrical. Definite masses were not recognized.

During the first two days in the hospital the patient complained of pains in the right hip and on both sides of the back and occasionally in the left shoulder. The temperature ranged between 103.2 and 99°F., with what was described as a sinus tachycardia. Chlortetracycline and Gantrisin therapy was continued until the sensitivities of the bacteria in the urine were reported, when chlortetracycline was discontinued and chloramphenicol started. These drugs did not seem to alter the course. The right-upper-quadrant pain and joint symptoms subsided. The back pain continued and was most prominent on the left side. Ten days after admission it was noted that "maximum tenderness is below the last rib on the left rather than in the left costovertebral angle." He continued to have a "constant nagging" pain in the back, on the left below the ribs. For a single day he had recurrent episodes of shooting pain in the left groin that had completely disappeared the next day. The tissues in the left flank became a little "boggy" and slightly edematous. Pain in the back on the left, fever and malaise continued.

FIGURE 1. *Lateral View of the Lumbar Spine, Showing Calcification within the Wall of the Abdominal Aorta. Arrows point to the calcification.*

On the fourteenth hospital day the abdomen was somewhat distended, and there was definite evidence of peritoneal irritation. Later during the same day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. OLIVER COPE*: "The blood pressure was 100 systolic, 70 diastolic." For a man with the history and physical findings of an arterial disease, this was a surprisingly low blood pressure; one wonders whether it was an isolated determination. This leads one to think of an adrenocortical accident; the persistently high temperature in spite of adequate chemotherapy is also consistent with acute adrenocortical insufficiency.

The laboratory data included the widely fluctuating white-cell counts, which, of course, fit with the fever and point to an acute infectious process. The Widal test, positive in a dilution of 1:160, I think, is considered borderline. Or is that completely negative for typhoid infection?

*Visiting surgeon, Massachusetts General Hospital.

DR. BENJAMIN CASTLEMAN: I believe it is negative.

DR. COPE: That the stool culture was negative for pathogens, I suppose, means negative for the salmonella group. Probably no other organism was sought unless the purpose of the culture was to search for a staphylococcus in view of the fact that the patient had had chemotherapy for some time. I doubt if the staphylococcus was considered because of the absence of diarrhea or other bowel symptoms. In addition the urine was cultured. I take it that the physicians were looking for infection in the left kidney or in the region of the left kidney such as a perinephritic abscess.

Was the amylase determined?

DR. MAURICE FREMONT-SMITH: It was normal.

DR. COPE: When, in relation to the onset, was the test made?

DR. FREMONT-SMITH: Two weeks after the onset.

DR. COPE: In pancreatitis, if it is measured as late as three weeks after onset, the amylase level may be back to normal. Was the eosinophil count done?

DR. CASTLEMAN: I do not think so.

DR. COPE: I take it that the chemical contents of the urine were not analyzed. Was the 17-ketosteroid level determined?

DR. CASTLEMAN: No.

DR. COPE: Before we see the x-ray films I shall rapidly go over the conditions that are on my mind. The first one that I think of, because of the amount of arterial disease and the previous occlusion, is dissecting aneurysm, with hemorrhage into the flank. There was, however, too much fever, and the curious skin flaking does not fit. So at the present moment I tend to dismiss that diagnosis.

A perinephritic abscess seems to be the most logical infectious process. From the clinical description at the end it is obvious that the inflammatory process was largely retroperitoneal with the symptoms and signs localized to the flank—tenderness, the development of edema and the transient but significant groin pain. Perinephritic abscess must have been in the minds of the examiners or certainly of the physician who wrote up this record because of repeated reference to the urinary findings.

Pancreatitis can fool us, but the absence of pain at onset is not the usual way for pancreatitis to start. On the other hand pancreatitis commonly gives rise to a retroperitoneal abscess as a late complication. The abscess occasionally points to the groin—gravitating out retroperitoneally that way.

The history of the ulcer, of course, may be a "red herring," but perforated ulcer, perhaps in the lesser peritoneal cavity extending down posteriorly on the left side, should also be considered.

I have alluded to the low blood pressure and the possibility of an adrenocortical hemorrhage. That occurred to me—I do not know just why. There was nothing in the original description of the patient to suggest an adrenal tumor; therefore, the hemorrhage would have occurred in a normal adrenal gland, and I find no inciting cause of such a hemorrhage.

Finally, if he had a dissecting aneurysm or a saccular aneurysm in the aorta, the aneurysm or blockage must have been below the renal artery because of the continued output of urine. May I ask Dr. Wyman to show us the x-ray films?

DR. STANLEY M. WYMAN: These films were selected from a number of examinations. The gall bladder is seen and does function although it is less dense than usual. The thoracic spine and ribs appear within normal limits except for the scoliosis, and the right upper femur appears quite normal. The heart shadow is prominent in the region of the left ventricle and the aorta is quite tortuous.

DR. COPE: Why is the left leaf of the diaphragm a little higher than the right? When was that film taken?

DR. WYMAN: I think it was taken early after admission to the hospital.

DR. COPE: Would you comment on that left leaf of the diaphragm?

DR. WYMAN: Fluoroscopic study was performed, and the diaphragm moved quite well at the time of the original examination. I saw the patient on two occasions. Later on, I believe, fluoroscopic study was made by another examiner, and I do not know how well the diaphragm moved then. I do not think there was a significant limitation of motion.

DR. FREMONT-SMITH: It moved normally.

DR. COPE: He had shoulder pain on the right before admission. Then he had acute shoulder pain on the left, which I interpreted as possibly referred pain from diaphragmatic irritation.

DR. WYMAN: The later films in this region were taken after the pain developed in the costovertebral angle and below the level of the eleventh rib. The psoas-muscle shadow on the right is quite well seen; the right-kidney shadow is well outlined. On the left the psoas-muscle shadow is less clearly visible, and the left-kidney shadow is not as well outlined; however, if one takes into consideration the amount of fecal material that is overlying it, that may play some part in the picture.

DR. COPE: What was the date of that film?

DR. WYMAN: It was taken two days later.

DR. COPE: Here the psoas-muscle and kidney shadows are slightly better outlined.

DR. WYMAN: That is correct. On the second examination, the left psoas-muscle shadow appears wider than the right. The kidney is fairly well outlined.

DR. COPE: The kidney is consistently lower on the left than on the right in both films. Does it look as if the left kidney were displaced downward?

DR. WYMAN: I should not go that far. The left kidney may be somewhat lower than it usually is, but

I think that position is perfectly compatible with a normal position in relation to the left leaf of the diaphragm. I do not think it is pushed down.

DR. COPE: You will not comment on the shadow except that it might be the overlying structures?

DR. WYMAN: I think it is probably the fundus of the stomach. The abdominal aorta, which is quite well seen, is densely calcified at this level.

DR. COPE: Do you see the aorta on the anteroposterior film?

DR. WYMAN: Not with certainty.

DR. COPE: I should not be surprised if this was a dissecting aneurysm, but I shall have to exclude the diagnosis because there was too much fever and too many signs localized in the left flank with evidence of retroperitoneal infection. I suspect the region of the left kidney, and the most logical diagnosis is a perinephritic abscess. There was no history of previous boils or infection, and I am uncertain what the flaking of the skin that developed later means. The flaking of the skin may have been present earlier, with some portal of entry of organisms that gave rise to a blood-borne infection. I am not satisfied with the organism that was found in the urine; *P. vulgaris* is not too uncommon in the urine. I am not at all sure that the patient had not had pancreatitis, with a gradual breakdown and a retroperitoneal abscess, which was the reason for the operation. Perforated ulcer cannot be excluded because anybody who has had an ulcer history in the past can have ulceration later, perhaps as a concomitant to infectious disease, that goes on to perforation. I should not be surprised if we found that to have been the origin of the retroperitoneal inflammation and abscess.

The last one is the hardest one to deal with—that is, the question of an adrenal hemorrhage. The major point that delays me is that the blood pressure, although low, was really not as low as it should have been with adrenal insufficiency; however, I am not sure that he had not had an adrenal hemorrhage, with resulting diaphragmatic and retroperitoneal complications.

DR. S. PETER SARRIS: Was a barium-enema examination done?

DR. WYMAN: It was noncontributory.

A PHYSICIAN: Was an electrocardiogram taken?

DR. FREMONT-SMITH: Not at this time.

A PHYSICIAN: Was there any bone disease?

DR. CASTLEMAN: No.

A PHYSICIAN: Was a pyelogram taken?

DR. CASTLEMAN: No.

A PHYSICIAN: Was retroperitoneal air injected to see if the mass could be outlined?

DR. CASTLEMAN: No.

A PHYSICIAN: What were the previous blood-pressure levels?

DR. FREMONT-SMITH: They were always normal.

A PHYSICIAN: Were blood cultures done?

DR. CASTLEMAN: Yes; they were all negative.

Dr. Fremont-Smith, would you tell us the opinion before operation?

DR. FREMONT-SMITH: The beginning of the illness was extraordinary; I do not think any of us can explain it. The patient had a sore throat and then he had a rash from his waist down that looked like a scarlet-fever rash. There was nothing above the waist. The throat looked pink and perfectly innocent, and the tongue was perfectly normal. Later on the rash came up over the abdomen and arms, and the peeling of the skin occurred later. The whole picture supported the diagnosis of a streptococcal infection. One could have called this scarlet fever except that he did not have a high initial fever or symptoms or signs of sore throat. He felt quite well. He came into my office and said that he had a sore gland under the left mandible. I could not feel this gland, and because I thought he was all right I permitted him to go to the country, where he had shaking chills and fever, with a temperature of 104°F. The local physician gave him many antibiotics and said he thought that there was some kidney trouble. The patient returned to Boston with a little pain in the left flank. I examined the urine and found a few white cells and 1 pus cast; I sent him to the hospital with the idea that he had a urinary infection. If Dr. Cope had been right beside the bed, I am sure he would have been as confused as he was standing here.

DR. FLETCHER H. COLBY: I think Dr. Cope is to be complimented on his summation of the facts that he has been given. He came nearer the diagnosis than we did. The fact that the patient was operated on by Dr. Arthur W. Allen and me showed the uncertainty that we had about making an accurate diagnosis in this case. The significant thing to us the morning of operation was that for the first time this man had evidence of a definite peritoneal irritation. We believed that he probably had retroperitoneal sepsis, perhaps a perinephritic abscess although there were certain findings that were against that. We were uncertain about what the sepsis was, but we were worried that it might rupture into the peritoneal cavity. We decided that now was the time to operate although we had postponed operation for quite a number of days.

When the tissues in the left flank were incised, definite edema of the subcutaneous tissue and the underlying muscle was found. The perirenal capsule was exposed and opened and was perfectly normal in every respect. The kidney was partially exposed—enough so that we were sure that there was no abnormality of the left kidney. The dissection was then carried down into the pelvis. Near the iliac vessels and near the psoas muscle we found a cavity in which there was thin, foul-smelling pus. We were satisfied that we had found the source of the elevated temperature but were uncertain about the exact nature of it.

When a finger was inserted in the cavity to put a drain down into the bottom of it, there was a tremendous gush of blood. In the bottom of the cavity there was some old blood clot. The hemorrhage was profuse, but it was easily controlled with a pack. When the pack was removed there was no further bleeding, but we did put Gelfoam in the bottom of the cavity and left a pack in.

DR. COPE: Was that below the kidney and in front of the renal fascia?

DR. COLBY: It was below the kidney and well below the renal fascia, immediately medial to the psoas muscle itself.

DR. CASTLEMAN: Come down and have a second chance, Dr. Cope.

DR. COPE: One would have to assume that the blood was the primary lesion, because otherwise I do not think it would have hemorrhaged at the bottom. The second thing is the sore throat, which Dr. Fremont-Smith described but was not mentioned in the record; that explains the origin of the temperature and the rash—the patient must have had streptococcal disease, and that was the secondary invader in a hematoma.

Where did the hematoma come from? It was presumably a vascular accident. The hematoma was below the kidney and did not, until the very last, interfere with the abdominal cavity and its contents; therefore, it did not come from one of the mesenteric vessels but was clearly retroperitoneal. It could have come from a branch of the aorta such as the lateral sacral artery, but presumably it came from the aorta itself below the inferior mesenteric artery, perhaps near the bifurcation. That would explain the changes in the legs. I believe, therefore, that this was a rupture of an aneurysmal sac in the lower end of the aorta.

CLINICAL DIAGNOSIS

Retroperitoneal sepsis, ?perinephritic abscess.

DR. OLIVER COPE'S DIAGNOSES

Before operation:
?Perinephritic abscess.
?Adrenal hemorrhage.
After operation:
Rupture of aneurysm of lower aorta.
Secondary streptococcal infection of hematoma.

ANATOMICAL DIAGNOSES

Streptococcal infection of arteriosclerotic aneurysm of abdominal aorta, with rupture into retroperitoneal space.

Arteriosclerotic aneurysms of iliac arteries, with occlusion of right.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: At the time of operation a culture was taken from the thin, purulent pus, and a pure culture of hemolytic streptococcus was obtained. The patient did not survive long after the operation.

DR. COLBY: He died that evening.

DR. CASTLEMAN: At autopsy we found that the kidneys were normal and that the cause of the hemorrhage was, as Dr. Cope suggested, rupture of an arteriosclerotic aneurysm of the abdominal aorta just above the bifurcation. Both iliac arteries had also arteriosclerotic aneurysmal dilatations; the right was completely occluded by an old organized thrombus (Fig. 2). The rupture occurred through the posterior wall of the aneurysm, close to the vertebral body. The aneurysm must have oozed slowly but was probably not completely perforated until the time of the operation.

FIGURE 2.

On microscopical examination the entire wall of the aneurysm and of the clot itself, which was old, was packed full of streptococci. The probable sequence of events was first an erysipelas or a scarlet-fever skin disease, with streptococcal infection that produced the bacteremia; then the organisms were caught in the arteriosclerotic aneurysm full of old blood clot, and the sepsis in the aneurysm led to the perforation. Further evidence for the same bacteremic process was found in another portion of the aorta. Just above the aortic valve were small arteriosclerotic plaques, with ulcerations and mural thrombi, which were also infected with streptococci.

A PHYSICIAN: Why was the blood culture negative?

DR. CASTLEMAN: That is not surprising in a patient who had received so much antibiotic treatment.

CASE 40332

PRESENTATION OF CASE

A sixty-three-year-old housewife was admitted to the hospital because of a "pulling sensation" of the right thigh.

The patient first noted "an uncomfortable pulling sensation" in the posterior aspect of the right thigh about eight months previously. The discomfort began after she sat for half an hour or so and was especially noted when she drove a car. It had never caused pain, caused her to limp or bothered her at night. There had been no noticeable increase in the size of the thigh. She denied trauma to this area.

Physical examination revealed a slightly obese woman lying comfortably in bed. Examination of the lungs, heart, abdomen and pelvis was negative. Just inferior to the right inguinal ligament was a firm, fixed, deep-seated mass, palpable from the middle of the anterior surface of the thigh medially to a point where it disappeared behind the adductor muscles. It was just palpable behind the hamstring muscles. The boundaries of the mass were indistinct, although the size was estimated at approximately 10 cm. in diameter. The subcutaneous veins of the thigh were more prominent over the femoral aspect of the mass. There was no change in temperature over the mass and no apparent pulsations. There was no edema of the skin of the extremity. There was no pain on motion of the right lower extremity except on compression of the mass from hyperflexion.

The temperature, respirations and pulse were normal. The blood pressure was 134 systolic, 76 diastolic.

Urinalysis was negative. Examination of the blood disclosed a hemoglobin of 13.2 gm. per 100 cc. The white-cell count was 6700, with a normal differential. The alkaline phosphatase was 1.6 units, the blood calcium 9.0 mg., and the phosphorus 3.2 mg. per 100 cc. A roentgenogram of the chest was normal. Roentgenograms of the pelvis and right hip showed slight demineralization of the pelvis; otherwise, no abnormality was noted in the pelvis or upper right femur. There was a soft-tissue mass, about 10 cm. in diameter, medial to the upper portion of the shaft of the right femur, just below the right inguinal ligament (Fig. 1). It was well circumscribed, rounded and less dense than the surrounding tissue. The superior portion showed areas of irregular streaks and patches of calcific density, some of which appeared ossified. In the lateral view the mass appeared separate from the bone.

FIGURE 1. *Anteroposterior View of the Upper Thigh. Note the large, well circumscribed, relatively radiolucent mass, which shows calcification of its proximal portion and along its periphery.*

On the third hospital day an operation was performed.

DIFFERENTIAL DIAGNOSIS

DR. GRANTLEY W. TAYLOR*: Since this is a tumor that is visible in the x-ray films, we might look at the films now.

DR. JOHN F. GIBBONS: There is a fairly well circumscribed mass medial to and overlying part of the upper femur. The lateral view shows that it is anterior to the femur. The density of the tissue that makes up this abnormal mass is less than that of the surrounding normal tissue. There is a collection of calcifica-

*Visiting surgeon, Massachusetts General Hospital.

tion, which is somewhat reticulate in character, at the top edge of the mass. The surrounding bones look normal. I do not see any other mass in the pelvis.

DR. TAYLOR: The x-ray report mentioned that some of the calcified material appeared to be ossified. Would you clarify that point?

DR. GIBBONS: I assume that the radiologist who interpreted the films thought that the architecture of the density in the upper portions of the mass resembled trabeculae and thus said it might be ossified rather than merely calcified. I am not at all sure of that.

DR. TAYLOR: The chest films were normal.

DR. GIBBONS: Yes.

DR. TAYLOR: The history is not profoundly significant. The main points are that for eight months this elderly woman had noted symptoms that were not very severe, that she never complained of pain but had an uncomfortable pulling sensation, that this discomfort had not occurred at night and that she had had no history of trauma. Physical examination revealed a tumor that was palpable in the thigh medially and extended around posteriorly. I think a short cut to diagnosis is to say that the tumor mass in the proximal thigh in this sixty-three-year-old woman was a localized disease, not a general disease, for she was in otherwise very good condition.

What are the possible and probable explanations for a tumor in this area that presents some calcification? I suppose that, if I am to make a broad sweep, I must say that tumors with areas of calcification may follow trauma, the conspicuous one being an ossifying or calcifying hematoma. After a hemorrhage of any sort calcium may be deposited in the area of clot. There was no trauma in this history. If she had trauma severe enough to cause hematoma resulting in this calcification I suppose that she would have recalled it. Also, calcification occurs with various low-grade traumas. Calcification is present in relation to bursas and to old arthritic processes about joints. Singularly enough, she had been free of any arthritic process in this area, and certainly there was nothing to suggest an antecedent or coincident bursitis, which might have accounted for this picture. It seems that I can dismiss trauma of any kind as a probable explanation.

I shall therefore consider the possibility of infection in the soft tissue. The first one I think of is tuberculosis. Calcification is seen in relation to old foci of tuberculosis. Certainly, nothing about this picture raises the question of an old, chronic tuberculous infection or of other types of infection that would give rise to a large mass, with calcification. Without elaborating further, I shall dismiss infection and concentrate on the problem of tumor in this patient.

A definite tumor, with some sensation that may have been perhaps due solely to this mass, was described. One condition that, perhaps loosely, comes into the field of tumor is aneurysm. Nothing was said about the pulses in the foot, and no pulsation was described in relation to the tumor; I think that, if we saw a similar tumor in an abdominal film and palpated a tumor in the abdomen, we should raise the question of an aneurysm in the abdominal aorta. However, I do not think an aneurysm of this extent in this area would be present without some associated findings either in the history or on physical examination.

I shall focus my attention on true tumors — benign and malignant. The duration, which was not long, does not preclude malignant tumors. If the tumor had been present for fifteen years I should be justified in saying that it was obviously benign or benign with recent malignant transformation. What sort of malignant tumors occur in elderly women? The commonest by all odds in metastatic carcinoma. Conspicuously, this was a large, single mass, with no other physical finding. It is most unusual for metastatic carcinoma to behave in this fashion. Moreover, I think if the patient had metastatic disease of this extent, there would have been some discoverable primary focus.

What primary malignant soft-tissue tumors appear in this area? Fibrosarcoma is probably the commonest. The age of the patient does not preclude fibrosarcoma although most of them occur in younger persons. Hemangiosarcomas are rare; they may have areas of calcification in them. Synovioma, a relatively rare malignant tumor, is usually found near a joint and frequently has in it areas of calcification of an irregular sort. I think that must be considered a possibility in this case.

Bone tumors of large extent in the vicinity of the hip are chondrosarcomas or osteochondrosarcomas and quite commonly occur in elderly patients, presumably as a transformation of a pre-existing benign chondroma. Since this mass conspicuously did not involve or implicate the bone, I can rule out chondrosarcoma as well as the other forms of primary bone tumor.

I come without too much circumlocution to the problem of benign tumors that may contain areas of calcification — fibromas, lipomas, neurofibromas and so forth. Almost any benign tumor may present areas of calcification as a result perhaps of minor hemorrhages with changes in the tissue. I do not think I can go farther than that in the general discussion. When I examine these x-ray films I note with interest that the tumor appears to be encapsulated or smoothly rounded, with definite margins, and that, in contradistinction, the parts of tumor not calcified have a higher radiolucency than the surrounding tissue. Radiologists have pointed out for years that a tumor with increased radiolucency contains fat. I think, therefore, that this patient very probably had a lipoma in the vicinity of the hip and that at some time or other there had been changes in it — perhaps hemorrhage, with the deposition of calcium. The operation was probably an exploration and biopsy. I should be inclined to suggest to such a patient that she keep such

a tumor, assuming the diagnosis of lipoma is correct, rather than subject her to an extensive and possibly damaging operative procedure.

DR. FLETCHER H. COLBY: Did this mass have the characteristic feeling of a lipoma?

DR. CLAUDE E. WELCH: The tumor felt very hard, as one might have guessed from the calcification. It was also extremely fixed; it was impossible to move it on the bone. The mass had displaced the femoral vessels medially, but the vessels appeared to be entirely normal. Pulsations distally were also normal.

DR. TAYLOR: Dr. Colby, about the consistence, I should say that a mass deep seated in a fat woman's buttocks would be very hard to characterize, other than to say that there was a mass.

DR. BENJAMIN CASTLEMAN: Dr. Welch, would you tell us your opinion before operation?

DR. WELCH: Dr. Wilma J. Canada, who looked at these x-rays films, told me that this was a benign lipoma, but our preoperative discussion concerned the possibility of a true lipoma or a liposarcoma. The relatively short duration of the symptoms, the fact that the mass was so firmly fixed and the rather irregular calcification throughout it led me to believe that it was possibly a liposarcoma. I am sure the main reason I made that preoperative diagnosis was that I had never been able to make it before and probably never would be able to make it again. Also, not sharing Dr. Taylor's reluctance to advise operation, I thought it best to remove the tumor. It came out without any undue difficulty and with no damage to any structure.

CLINICAL DIAGNOSIS

Liposarcoma of thigh.

DR. GRANTLEY W. TAYLOR'S DIAGNOSIS

Lipoma of thigh.

ANATOMICAL DIAGNOSIS

Lipoma of thigh, with metaplastic ossification.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: The tumor, as predicted, was made up primarily of fat, with many calcified areas scattered throughout. Many of these areas were red, and we thought some of them might be not only bone but actually marrow. Microscopical examination showed that this was a benign lipoma with foci of true bone scattered throughout the tumor and also good cartilage in places. There was no evidence of marrow. Some pathologists, when finding fat, bone and cartilage, might term the tumor a benign mesenchymoma since more than one mesenchymal element is present. I prefer to call it a lipoma, with foci of metaplastic bone and cartilage formation. This may occur whenever there is destruction of tissue and young connective tissue is forming and, in turn, developing into bone and cartilage. Dr. Petersen took some interesting x-ray films of the specimen; will you demonstrate them, Dr. Gibbons?

DR. GIBBONS: In the films of the specimen one can be more sure that there are actually bone trabeculae. This film (Fig. 2) was made with the tumor enclosed in a bag filled with water to show the decreased density of the fat compared to the density of water, which is equivalent to normal tissue density; it also enhances the appearance of the calcification.

FIGURE 2. *X-Ray Examination of the Specimen, Which Has Been Placed in Water, Showing Its Decreased Density Compared with Water, Indicating the Presence of Fat.*

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PUBLIC HEALTH IN CHRONIC DISEASE

FOR his Cutter Lecture, published in this issue of the *Journal*, Dr. Thomas Parran chose the subject "Contributions of Public Health to the Control of Chronic Disease." He is particularly qualified to treat this topic, for as Surgeon General of the United States Public Health Service for twelve years he has administered an agency that has probably had as much to do with the control of chronic disease as any institution that could be named.

Commissioned an officer in the United States Public Health Service in 1917, Dr. Parran remained in service with the Government until his appointment as Health Commissioner of the State of New York in 1930. In 1936 he returned to the Public Health Service as Surgeon General and remained in that post until 1948, when he became dean of the University of Pittsburgh Graduate School of Public Health. He has represented the United States on the interim commission of the World Health Organization and on the directing council of the Pan American Sanitary Bureau. He is a past president of the American Public Health Association, a trustee of the Rockefeller Foundation and a member of numerous other national and international professional organizations. He is the author of *Shadow on the Land* (1937) and *Plain Words about Venereal Diseases* (1941).

In this seventieth Cutter Lecture Dr. Parran recognizes chronic disease for what it is: the cumulative aftermath of most, if not all, of the serious afflictions known to medical science. He therefore wisely stresses the impossibility of studying chronic disease as a unit. To do so would be like studying the "tides of Massachusetts Bay as an isolated phenomenon unrelated to the larger planetary, gravitational and astronomic forces that produce them." In these words Dr. Parran paid tribute to an illustrious name in Boston, that of Dr. Oliver Wendell Holmes. In a lecture to the Massachusetts Historical Society delivered before the Lowell Institute on January 29, 1869, Dr. Holmes* remarked:

> **A geographer who was asked to describe the tides of Massachusetts Bay, would have to recognize the circumstance that they are a limited manifestation of a great oceanic movement. To consider them apart from this, would be to localize a planetary phenomenon, and to provincialize a law of the universe.**

What was agreeable rhetoric in 1869, requiring no more than an unspoken acquiescence on the part of an audience nodding under the gaslight, becomes an awesome reflection in 1954. Today, when there seems to be so little time for nodding, Dr. Parran has given fresh life to the words of the little professor, emphasizing that "The problem of public health is even more difficult than the study of tides since one is dealing neither with recurring nor with static phenomena, but with dynamic change, with nonrecurring movement as science progresses and social concepts broaden."

*Holmes, O. W. Medical profession in Massachusetts. In *Medical Essays 1842-1882*. 445 pp. Boston: Houghton Mifflin, 1883. Pp. 312-369.

OPERATION ALERT

THE Massachusetts Civil Defense Agency, under the expert direction of Colonel John J. Maginnis, has published in a July issue of *Civil Defense News* an analysis of its own part in the first nation-wide practice alert, held on June 14 and 15.

Public participation and co-operation were gratifying, being considered nearly 100 per cent satisfactory. The results otherwise, however, although conspicuously good so far as willingness and effort are concerned, are considered as representing only a good start toward the ultimate degree of organization that must be achieved.

Improvement, according to this analysis, is necessary in the simplification and streamlining of operations at control centers, in a better inventory of available personnel and supplies and their location, and in plans for their speedier movement. There is also need for a quick appraisal of what remains of an original inventory after a bomb burst.

A well organized civil-defense setup in towns and cities adjacent to a bombed area was shown to be particularly important, especially in the municipalities of the 15-mile and 20-mile radiuses. Greatly increased personnel, better warning signals and much more effective and detailed organization are needed in the communities of these areas, for they are the ones from which first aid must be forthcoming. They are the hills unto which the eyes of the stricken city will first be cast, and must answer the question, "Whence cometh my help?"

The most important question that remains to be answered is what the inhabitants of a forewarned city are to do in the event of an expected attack; whether to evacuate or seek shelter. Obviously, the time available for action is the important factor in making such a decision, but clear instructions on this point must sooner or later be issued, in unmistakable terms.

ARMY MEDICAL EDUCATION

ACTING on a request from the Army Medical Service, the National Research Council of the National Academy of Sciences organized in June a new committee on Army Medical Education. This committee, replacing the former Advisory Committee of the Army Medical Service Graduate School, will include in the scope of its activities the education and training problems of the entire Army Medical Service, as well as serving in an advisory capacity to the surgeon general.

Thoroughly representative, the committee includes in its membership Dr. Dean A. Clark, general director of the Massachusetts General Hospital, as chairman; Dr. Joseph M. Hayman, Jr., dean of Tufts College Medical School; Dr. Franklin C. McLean, University of Chicago; Dr. John H. Mulholland, Department of Surgery, University of Pennsylvania; Dr. Grant Taylor, Graduate School of Medicine, University of Texas; Dr. Gaylord W. Anderson, University of Minnesota; and Dr. Tom Bradley, National Research Council, as executive secretary.

The committee will serve three important functions:

To determine, analyze and evaluate those professional and technical fields where civilian developments must be supplemented to meet the requirements of national defense.

To review the professional and technical programs in the Army Medical Service, to determine whether or not they adequately meet the professional and technical needs for defense.

To review educational methods and career patterns being employed by the Army Medical Service, to determine whether they are designed to produce the leadership and professional requirements for defense.

The formation of such a committee for such purposes indicates an alert and progressive Army Medical Service, aware of its responsibilities and the broad front on which they must be met.

TOBACCO COMPANY AIDS RESEARCH

THE National Institutes of Health, research agency of the Public Health Service of the United States Department of Health, Education and Welfare, is in receipt of an electron accelerator, the most powerful of its type. The 3,000,000-volt Van de Graaff generator, the gift of the Liggett and Myers Tobacco Company, was built in Cambridge, Massachusetts, in 1950, for an experimental program of the tobacco company, the nature of which was not stated in the release emanating from the Institutes.

The 30-ton accelerator, which has been installed in the radiation wing of the 500-bed Clinical Center at the National Institutes of Health in Bethesda, Maryland, was accepted for the Government last March by Secretary Oveta Culp Hobby. It will be used, according to the release, for research on the radiochemical and biologic effects of x-rays and electron energy, and can also be modified to produce short-lived radioactive isotopes.

There is less activity at present in the publication of medical books, than usual. Most of the re-publications of late are minor productions. We trust, however, that something excellent may be forthcoming for long winter evenings.

Boston M. & S. J., *August 23, 1854*

CORRESPONDENCE

PSYCHONEUROSIS OR ORGANIC LESION?

To the Editor: Psychiatry has done much that is good as well as much that is harmful. Physicians now have had enough experience to know that medical science cannot always differentiate the symptoms that are interpreted as subjective psychoneurotic complaints and those that may later become objective. The early symptoms of doubtful organic origin are often indicative later of real organic disease, and this point should be emphasized in teaching programs.

SARAH PARKER WHITE, M.D.

Boston

SPECIALISTS AND GENERALISTS

To the Editor: I believe that the article by Dr. J. H. Means on the "Choice of Opportunities in Medicine" in the May 6 issue of the *Journal* contains some misunderstandings that need correction, as does a similar article of his published in the March issue of the *American Practitioner.*

To me it is regrettable that every young student who graduates from medical school is called a physician and surgeon, though actually he is a general practitioner (generalist). This fact is recognized in Europe, where the practitioner is denied the right to call himself a specialist in medicine (internist) or in surgery (surgeon).

If a medical graduate does not go into practice immediately but continues further special work in the field of medicine exclusively he may become a specialist in medicine —or similarly in the field of surgery—and upon entering practice may be expected to abstain from work in all other fields. As an internist he may further restrict his field to one of the subspecialties, but I believe that this is not possible without a thorough knowledge of internal medicine as a whole, and that this applies equally to the subspecialties in surgery.

There is a large field of work available for the specialist of medical diseases, as he can solve the majority of the diagnostic and therapeutic problems that confront the general practitioner.

It is unfortunate that the value of the general practitioner and his important place have not been recognized, and it is just as bad for a practitioner to consider himself a specialist in medicine without proper qualifications.

I think that the word "internist," an unfortunate acquisition from Germany, should be replaced by the designation "specialist in medicine." The official German title of such a specialist is "Facharzt für innere Krankheiten," pertaining to internal ailments only, exclusive of skin, eyes and so forth.

Dr. Means's difficulty in outlining the place of an internist can be understood partly because he has probably had no contact with socialized medicine. Under such a regime the role of a general practitioner is that of a highly trained medical clerk who is compelled to refer patients with serious or prolonged internal diseases to an internist.

H. B. Eisenstadt, M.D.

Port Arthur, Texas

Note: The graduate fresh from medical school is a physician in the broad sense of the word; he is certainly not ready to be called, nor is he usually called, a specialist in medicine or a surgeon or, indeed, any kind of practitioner until he has had considerably more training. — Ed.

A NEW COMPLICATION OF LAST TRIMESTER OF PREGNANCY

To the Editor: Since the end of World War II, it has become increasingly apparent that many private obstetric patients have an added anxiety, which stems from the uncertainty of the exact date of delivery. The increasing need for more certain information concerning the exact date relates to the professional convenience of the practical nurse whom resourceful patients have tentatively engaged to return home with them after the greatly foreshortened lying-in period in the hospital.

Obstetricians are no longer surprised that patients discuss practical nurses at the time of the first prenatal visit, usually several weeks after a missed period. It is a surmise, and yet possible, that the missed period is planned in relation to a specific nurse's availability.

As term approaches, the tensions previously alluded to are intensified by frequent telephone calls and even visitations from the practical nurse acquainting the prospective parturient patient with the tight schedule. If nature, most unkindly, does not prove helpful on the due date conscientiously calculated by the obstetrician seven or eight months previously, the particular nurse's commitments to the patient are, in many cases, terminated. Thus, the anxiety of the expectant mother is realized—she and the newborn infant are to be set afloat without the aid and direction of the solid, all-knowing and helpful skipper at the helm.

The obstetrician, once made aware of this disappointment and frustration, will strike out to prevent the dilemma by attempting induction of labor, assuming that all requirements for success are present—namely, the "ripened" cervix, engaged presenting part and absence of cephalopelvic disproportion. The obstetrician will often do so even at the cost of greater inconvenience to himself. Even then, nature may prove itself reluctant, and the patient is forced to retreat from the hospital with added frustration, not to mention added expense.

The situation deteriorates further when the patient thinks of the need for calling on relatives or friends whose response may be lukewarm or absent.

At this point, the obstetrician attempts to bridge the gap by artful reassurance and ego-building technics, in the meantime wearing out his secretary by calling all the practical nurses on his list.

If the nursing situation has not been resolved on the final day of parturition, the patient is left bereft of excitement after the first post-partum day. She again realizes her responsibilities and magnifies her own inadequacies, unaware that for generations babies have been born and reared without this added luxury, which for so many has assumed an exalted place in contemporary culture.

The concept of this complication in late pregnancy was emphasized when a patient informed me recently that her baby must be delivered one week later than the anticipated date, for the practical nurse she hoped to have would not be available until then.

If the psychiatrists are correct, how "rejected" the premature and post-mature infants must be!

Archie A. Abrams, M.D.

Boston

BOOK REVIEW

Country Doctor: C. W. Larison, 1837-1910. Physician, farmer, educator, reformer. By Harry B. Weiss, Sc. D. 8°, paper, 235 pp., with illustrations. Trenton, New Jersey: New Jersey Agricultural Society. $2.50.

This is the story of a rural physician who was a power in his community and had many interests aside from medicine. He first attempted to study medicine at the Pennsylvania College of Medicine in 1855, but gave up at the end of the first term because he believed that he needed further information about languages, mathematics, philosophy, literature, natural history and other subjects. In 1861 he entered the Geneva Medical College, receiving his medical degree in 1863.

During the period between the two medical schools he went to high school and college, taught school, began a nursery business and planted 6000 peach trees on his farm. He opened a private school in 1860 for specially chosen pupils.

As a physician he was the typical country doctor of the period, beloved by all. He was the founder of the Ringoes Baptist Church in 1867. In 1874 he became professor of natural history at the University of Lewisburg, Pennsylvania, at an annual salary of $1400. This appointment was probably influenced by the reputation that he had acquired in the subject and by his experience in teaching in his private school, at the Ringoes Seminary, formerly the Amwell Academy, and as principal of the Flemington High School. In 1875 he founded the Academy of Science and Art in which natural science, mathematics, Latin, English, drawing, music and elocution were taught. He was a firm believer in the study of nature and geography in the field, and his students were taken on long and short trips over the countryside.

As early as 1856 Dr. Larison became interested in spelling reform and gave much time to the subject; in 1881 he wrote his *Elements of Orthoepy* and in 1883 *The Fonic Speller and Sylabater.* In 1885 the Fonic Publishing House came into being, and thereafter all his publications were issued from this press, which he owned and operated. The *Journal of American Orthoepy,* later the *Journal of Orthoepy and Orthography* published in Ringoes, New Jersey, first appeared in January, 1884. The *Journal* came to

an end with the December, 1909, issue after an existence of twenty-six years. Four ephemeral magazines were published. As soon as one failed he began another. *Ringoes* was a monthly, devoted to the history of Ringoes, and the first number was issued in August, 1889. It ceased publication with the twelfth issue, in March, 1890. *The Journal of Health* (Ringoes, New Jersey), which first appeared in January, 1891, ended its career in December, 1892. The *Journal* was followed in April, 1892, by *The Spelling Reformer*, also issued from Ringoes, for 12 numbers, giving up the ghost in March, 1893.

While his *Journal* was expiring, Larison was planning another, *Amwel: past and present*, also printed at Ringoes in phonic orthography. The first number (January, 1910) was the only one published, the journal ceasing because of the author's death. He was always short of cash and died a poor man.

Larison's bibliography is interesting, consisting of 5 periodicals and 24 books and pamphlets. Thus ends the story of a many-sided man, a successful, respected and beloved physician, who had outside interests. His overwhelming desire to reform spelling was prefigured as far back as Caxton (1490), Ben Jonson (1605), Jonathan Swift (1712), Thomas Spencer (1750), Benjamin Franklin (about 1760) and Isaac Pitman and A. J. Ellis, who devised their phonotypic alphabet in 1847.

JAMES F. BALLARD

BOOK RECEIVED

The receipt of the following book is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be recieved as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Causality in Natural Science. By Victor F. Lenzen, Ph.D., professor of physics, University of California, Berkeley, California. 8°, cloth, 121 pp. Springfield, Illinois: Charles C Thomas, 1953. $3.00. (Publication No. 213, *American Lecture Series*.)

This discussion of causality is extensive in scope, and there is a chapter on its applicability to biology. There is a bibliography of 91 titles and an index.

NOTICES

NATIONAL RESEARCH COUNCIL FELLOWSHIPS

Applications are being accepted by the Division of Medical Sciences of the National Research Council for postdoctoral research. The following program is announced for 1955-1956:

Fellowships in cancer research awarded by the American Cancer Society for study in all branches of the biologic, chemical and physical sciences (citizens of the United States are eligible).

British-American exchange fellowships in cancer research awarded by the American Cancer Society to citizens of the United States for advanced study in Great Britain and by the British Empire Cancer Campaign to British subjects for research in the United States.

Fellowships in the medical sciences supported by the Rockefeller Foundation and Lilly Research Laboratories for research in the basic medical sciences (Rockefeller fellowships are open to citizens of the United States and Canada; Lilly fellowships are open only to citizens of the United States).

Fellowships in tuberculosis granted by the National Tuberculosis Association (citizens of the United States who are graduates of American schools are eligible).

Fellowships in radiologic (diagnostic) research supported by the James Picker Foundation (appointments are not limited to citizens of the United States).

Applications for any of these programs must be submitted before December 10, 1954. Information may be obtained from the Fellowship Office, National Academy of Sciences—National Research Council, 2101 Constitution Avenue, N.W., Washington 25, D.C.

SOCIETY MEETINGS AND CONFERENCES

AUGUST 24. Consultation Clinic for Crippled Children in Massachusetts. Page 1054, issue of June 17.

AUGUST 17–20. Argentine Congress of Thoracic Surgery. Page 708, issue of April 22.

AUGUST 24. Course in Care of Atomic Casualties. Page 285, issue of August 12.

SEPTEMBER, 1954–MAY, 1955. Venereal Disease Research Laboratory Courses. Page 285, issue of August 12.

SEPTEMBER 6–10. International Poliomyelitis Congress. Page 399, issue of March 4.

SEPTEMBER 6–11. University of Illinois College of Medicine. Annual Assembly in Otolaryngology. Page 1092, issue of June 24.

SEPTEMBER 6–11. American Congress of Physical Medicine and Rehabilitation. Page 246, issue of August 5.

SEPTEMBER 7–10. Nineteenth Annual Congress of the United States and Canadian Sections of the International College of Surgeons. Page 828, issue of November 12.

SEPTEMBER 12–15. Second International Congress of Cardiology. Washington, D. C.

SEPTEMBER 13–15. American Association of Blood Banks. Page 968, issue of June 3.

SEPTEMBER 16–18. American Heart Association. Annual Scientific Sessions. Washington, D. C.

SEPTEMBER 22–24. Mississippi Valley Medical Society. Page 246, issue of August 5.

SEPTEMBER 22. New England Pediatric Society. Page 968, issue of June 3.

SEPTEMBER 24. American Medical Writers' Association. Page 246, issue of August 5.

SEPTEMBER 30–OCTOBER 2. Canadian Society for the Study of Fertility. Page 285, issue of August 12.

OCTOBER 1 and DECEMBER 3, 1954; FEBRUARY 4 and APRIL 1, 1955. New England Society of Anesthesiologists. Page 1034, issue of June 17.

OCTOBER 7 and 8. Pan-Pacific Surgical Association. Page 1092, issue of June 24.

OCTOBER 8 and 9. Academy of Psychosomatic Medicine. Page 748, issue of April 29.

OCTOBER 11–15. American College of Physicians Course. Page 44, issue of July 1.

OCTOBER 16–22. American Occupational Therapy Association. Page 1092, issue of June 24.

OCTOBER 18–29. New York Academy of Medicine Graduate Fortnight. Page 44, issue of July 1.

OCTOBER 25–27. New England Postgraduate Assembly. Hotel Statler, Boston.

OCTOBER 25–30. Course in Care of Atomic Casualties. Page 285, issue of August 12.

OCTOBER 27. New England Obstetrical and Gynecological Society. Annual Meeting. Hotel Somerset, Boston.

OCTOBER 27–29. International Symposium on the Hypophyseal Growth Hormone. Page 1092, issue of June 24.

OCTOBER 31–NOVEMBER 4. American Institute of Dental Medicine. Page 968, issue of June 3.

NOVEMBER, 1954–MAY, 1955. Sanitary Engineering Center Training Programs. Page 122, issue of July 15.

NOVEMBER 3–5. National Society for Crippled Children and Adults. Page 44, issue of July 1.

NOVEMBER 12 and 13. Inter-Society Cytology Council. Page 968, issue of June 3.

NOVEMBER 15–26. Laboratory Diagnosis of Tuberculosis. (Course.) Page 246, issue of August 5.

DECEMBER 5. American Academy of Dental Medicine. Page 1092, issue of June 24.

DECEMBER 8. New England Pediatric Society. Page 968, issue of June 3.

DECEMBER 13–17. Congress of Obstetrics and Gynecology. Page 748, issue of April 29.

JANUARY 10–15, 1955. Course in Care of Atomic Casualties. Page 285, issue of August 12.

MARCH 7–12, 1955. Course in Care of Atomic Casualties. Page 285, issue of August 12.

MAY 4 and 5, 1955. American Psychosomatic Society. Page 286, issue of August 12.

JUNE 21–25, 1955. National Association for the Prevention of Tuberculosis. Page 540, issue of March 25.

CALENDAR FOR THE WEEK BEGINNING THURSDAY, AUGUST 26

THURSDAY, AUGUST 26

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*9:00–10:00 a.m. Arthritis Grand Rounds. Robert Breck Brigham Hospital.

10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*11:00 a.m.–12:30 p.m. Hand Clinic (Physical Medicine and Rehabilitation Service). Boston City Hospital.

*11:00 a.m.–12:00 m. Pediatric Conference. Mount Auburn Hospital, Cambridge.

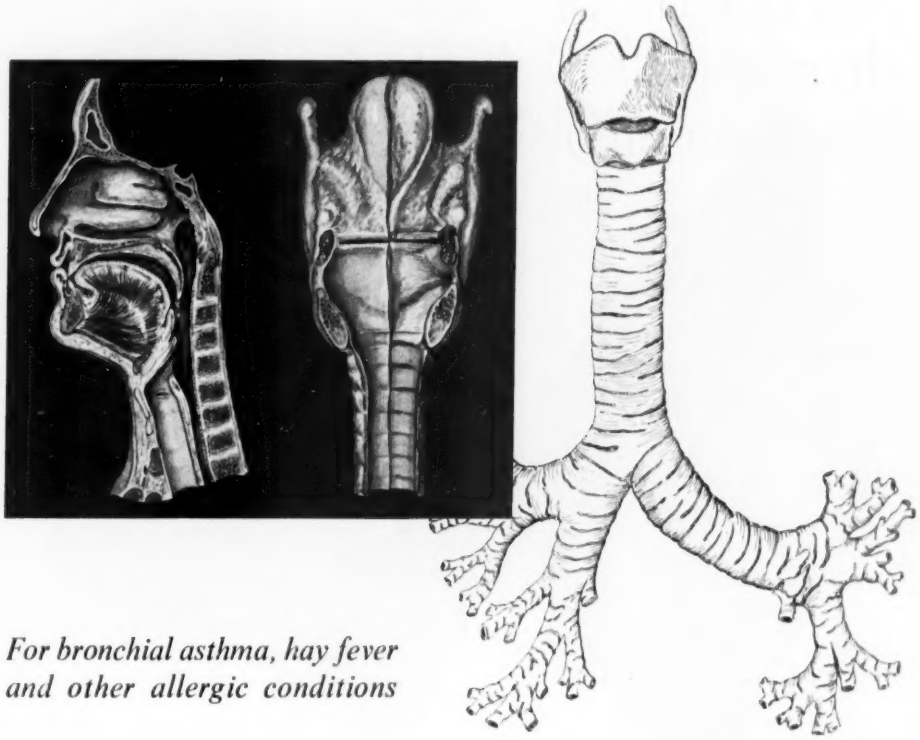
*11:00 a.m. Thoracic Conference. Unusual Types of Diaphragmatic Hernia. Dr. Hugh Wilson. Main Amphitheater, Peter Bent Brigham Hospital.

FRIDAY, AUGUST 27

*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

*9:30–11:30 a.m. Medical and Surgical Grand Rounds. Drs. George W. Thorn and Francis D. Moore. Main Amphitheater, Peter Bent Brigham Hospital.

(Notices concluded on page xxv)



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different because

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Formula: Each 'Daprisal' tablet contains 'Dexedrine' Sulfate (dextro-amphetamine sulfate, S.K.F.), 5 mg.; amobarbital, ½ gr. (32 mg.); acetylsalicylic acid, 2½ gr. (0.16 Gm.); phenacetin, 2½ gr. (0.16 Gm.).

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AGE	SEX	Dura... Symptoms before ACTH	...tal Dose (units)	Days of Treatment	Initial Improve... ment	...nitial ...nprove- ment	Complete Relief	Remar...
11	F	24 hrs.	200	3	48 hrs	48 hrs.	96 hrs.	Gay &
26	M	48 hrs.	125	3	48 hrs	48 hrs.	96 hrs.	Gay &
48	F	96 hrs.	60	2	24 h...	...4 hrs.	72 hrs.	Gay &
	M	72 hrs.	200	3	24...	... hrs.	48 hrs.	Falk, Allen...
	M	5 days	180	3		...	3½ days	& Bennet...

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Three days of treatment, implying a small total dose and economy, suffice as a rule.

References: 1. Flood, J. H.: Bull. Guthrie Clinic 21: 3, 1951. 2. Gay, L. N., and Murgatroyd, G. W., Jr.: J. Allergy 23: 215, 1952. 3. Falk, M. S., et al.: J. Invest. Dermat. 18: 307, 1952.

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RADIOLOGIST, Board Certified, seeks part-time association in hospital or office practice in Boston or vicinity. Also available for locum tenens and weekend coverage. Address A95, *New Eng. J. Med.* 8-3t

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X-RAY TECHNICIAN desires position in hospital. 5 years' experience in both hospitals and private offices. Address A110, *New Eng. J. Med.* 8-1t

WANTED—Monthly accounts, preferably to be done at my home. Address A109, *New Eng. J. Med.* 8-2t

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29-YEAR-OLD PHYSICIAN awaiting residency in orthopedics wishes temporary position from October 1, 1954 to July 1, 1955. Experience: 3 years' university surgery training, plus 2 years' Navy general surgery and orthopedics. Address A108, *New Eng. J. Med.* 7-2t

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(Advertisements continued on page xxiv)

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Pediatric ERYTHROCIN combats infectious cocci *within 2 hours* after it's administered—maintains inhibitory levels for 8 hours. It's highly active against the cocci—including those coccal organisms that have become resistant to penicillin and other antibiotics.

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DOSAGE...
One 5-cc. teaspoonful represents 100 mg. of ERYTHROCIN

25-lb. child ½ teaspoonful
50-lb. child 1 teaspoonful
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Every 4 to 6 Hours

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For the study, care and treatment of emotional, mental, personality and habit disorders.

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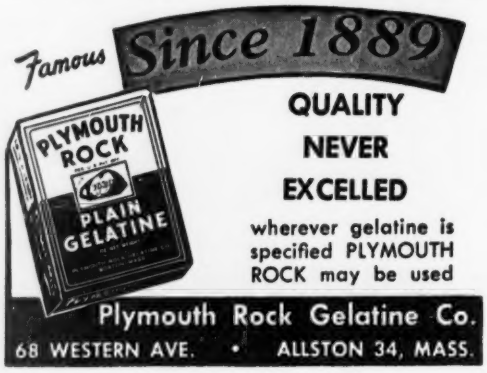

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NOTICES *(Concluded from page 322)*

*10:00 a.m. Tuberculosis Surgical Clinic. South End Health Unit, 57 East Concord Street.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*1:30 p.m. Fertility and Endocrine Clinic. Free Hospital for Women, Brookline.
*1:30 p.m. Tumor Clinic. Mount Auburn Hospital, Cambridge.
*4:00–10:00 p.m. Alcoholism Clinic. By appointment. Washingtonian Hospital, 41 Waltham Street.

SATURDAY, AUGUST 28
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*8:30–10:00 a.m. Orthopedic Staff Conference, Boston City Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.

MONDAY, AUGUST 30
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*8:00–9:00 a.m. Medical and Surgical Clinic on Diabetic Problems. New England Deaconess Hospital.
*8:30–9:30 a.m. Clinic by entire Surgical Staff and Anesthesia Staff. Cheever Amphitheater, Dowling Building, Boston City Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*12:15–1:15 p.m. Clinicopathological Conference. Main Amphitheater, Peter Bent Brigham Hospital.
*1:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.
5:15 p.m. Staff meeting. Harris Hall, New England Deaconess Hospital.

TUESDAY, AUGUST 31
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*9:00 a.m. Geriatrics Clinic. Peter Bent Brigham Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*12:00 m. Pediatric Grand Rounds. Cheever Amphitheater, Dowling Building, Boston City Hospital.
*12:15–1:15 p.m. X-Ray Conference. Dr. Merrill Sosman. Main Amphitheater, Peter Bent Brigham Hospital.
12:30–1:30 p.m. Medical Journal Club. Joslin Clinic. New England Deaconess Hospital.
*5:00–6:00 p.m. Service Meeting Followed by Clinical Conference (Medical, Surgical, Obstetrical). Faulkner Hospital.

WEDNESDAY, SEPTEMBER 1
*8:00–8:45 a.m. Case Presentations. Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
10:30–11:15 a.m. Lecture on Diabetes for Doctors and Patients by a Member of the Joslin Clinic. Joslin Auditorium, New England Deaconess Hospital.
*11:00 a.m. Boston State Hospital Psychiatric Seminar. Reception Building Auditorium, 591 Morton Street, Dorchester.
*12:00 m.–1:00 p.m. Clinicopathological Conference. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.
*12:30 p.m. Weekly Pathological Meeting. Joslin Auditorium, New England Deaconess Hospital.
*12:30–1:30 p.m. Weekly Staff Review of Autopsies. Dr. William A. Meissner. Joslin Auditorium, New England Deaconess Hospital.
*1:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.
*1:00 p.m. Massachusetts General Hospital Hand Clinic. Out-Patient Department.
*2:00–3:00 p.m. Pediatric Conference for Practitioners. Jimmy Fund Building, Children's Medical Center, 35 Binney Street.
*4:00–5:30 p.m. Fracture Lecture. Boston City Hospital.
*4:00–6:00 p.m. Overholt Thoracic Conference. Joslin Auditorium, New England Deaconess Hospital.
*6:00 p.m. Alcoholism Clinic. Peter Bent Brigham Hospital.

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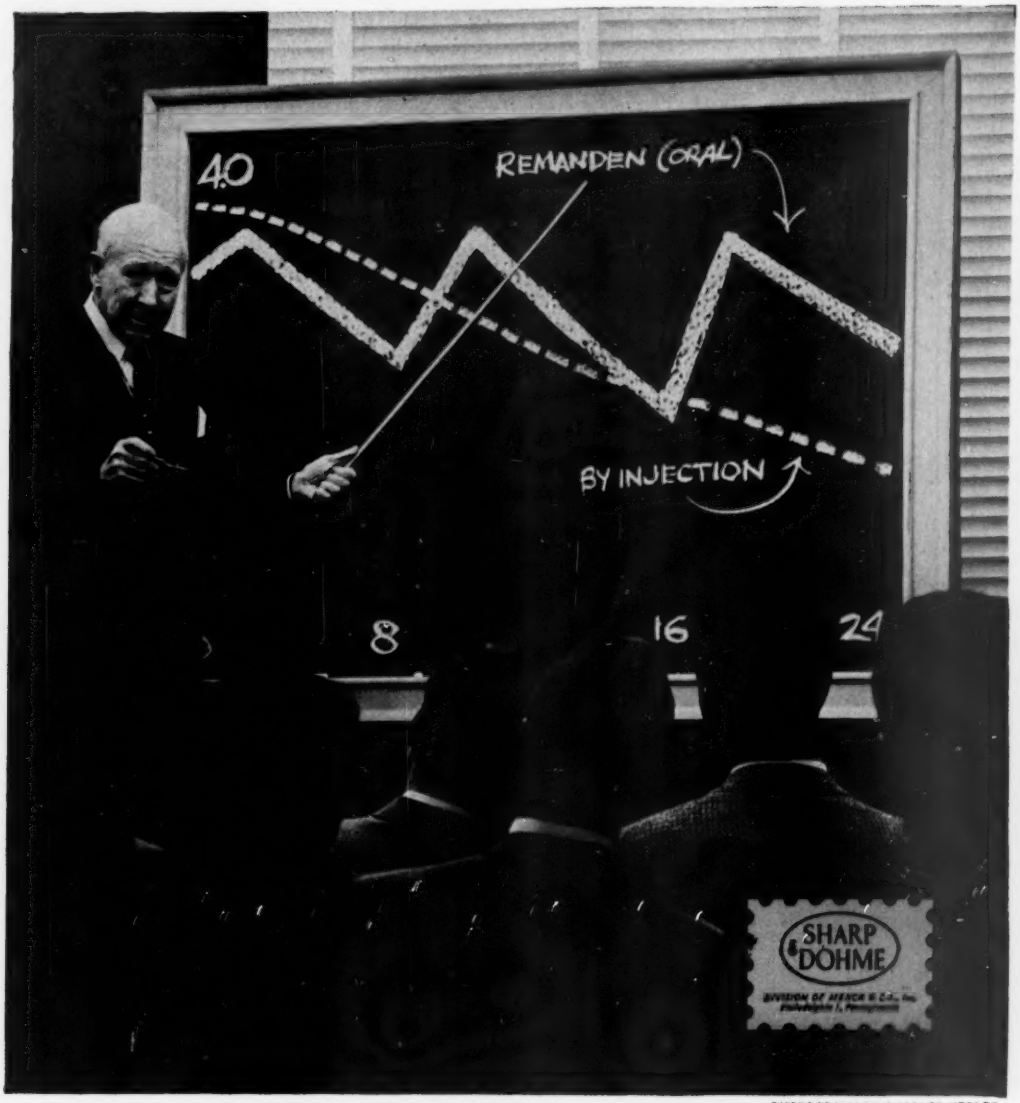

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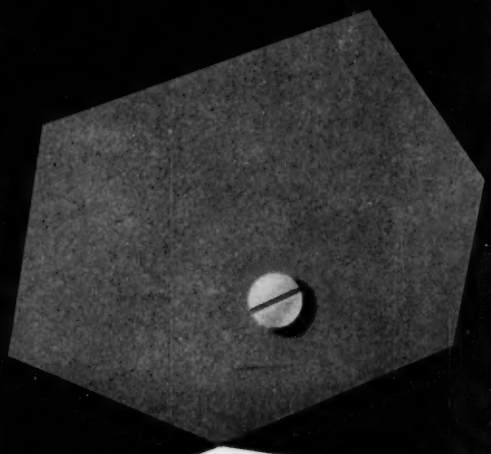

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